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PHYSIOLOGY OF THE LARYNX. A RESUME AND DISCUSSION OF THE LITERATURE FOR 1938.

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Negus,[1] in an especially brilliant article, discusses the evolution of the speech organs in man, and includes much of importance from the physiological standpoint. He points out that "human speech depends primarily on the production of sound in the larynx, an organ which corresponds to the reed in various musical instruments, or to the sound box of the gramophone, which sound is modified by resonators above and below the glottis. It is further modified by various "stops" which interrupt the vibrating column of air, or direct it through either the mouth or the nose. These "stops" are the vocal cords, ventricular bands, soft palate and nasopharyngeal sphincter, the tongue, the fauces and the lips. Negus adds, "if air is expelled from the lungs with the glottis partially closed, the vocal cords are thrown into vibration; the air current is thereby divided into a series of puffs and sound is produced."

Thus, Negus places the human voice in the category of a "reed" instrument, such as many of the modern woodwinds, and removes it from the classification of a vibrating string instrument. This concept is supported by G. Oscar Russell, whose work is discussed in an interesting resumé by Schatz.[2] Russell, as a result of rather convincing experiments with the laryngoperiscope, rapid exposure X-rays, oscillographs, and stroboscopic observations upon the vocal cords, in conjunction with co-ordinated sound-producing mechanisms and photo-

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graphic records of his subjects, has concluded, as does Negus, that "the vocal cords produce sound not by vibrations, as do strings in a musical instrument, but by repeated puffs or explosions of air released from the trachea by the rapid opening and closing of the valve-like action of the vocal cords . . . they function just as do our lips when playing the trumpet or cornet." Conclusions drawn indicated that variations in pitch are not produced by changes in merely the length of the vocal cords (which would be the case if they were vibrating strings).

This concept is most important and interesting. It finds further support in the writings of Guthrie,[13] who says, "from time to time, the larynx has been compared to various types of musical instruments. It can in no wise be likened to a string instrument as alterations of pitch do not depend upon changes in the length of the cords. To some extent, the larynx resembles a reed or a siren — what actually happens is that the vocal cords forming the glottic margins are approximated and hardened by muscular action; then by the increasing pressure of air from the lungs the glottis is opened and some air escapes. By reason of the elasticity of its margins, the glottis again closes, and by the rapid succession of these movements of opening and closing, a note is produced, the pitch of which depends upon the number of glottic movements a second. The simile of the siren holds good, in that the sound is the result of a rapid series of puffs of air. But the number of movements a second depends, in the larynx, upon the rigidity, thickness and tension of the vocal cords rather than upon the air pressure. . . . Sir Richard Paget has aptly compared the vocal cords to the lips of a trumpeter."

Wessely[21] maintains a similar attitude and expresses the opinion that the pressure of expired air causes an opening of the glottis, which in turn periodically interrupts this column of expired air. This becomes possible through changes taking place in the tonus of muscles controlling the labia vocalia, which changes can take place exceedingly rapidly.

[Negus, Russell and Guthrie represent perhaps the most profound thinkers, and ablest experimenters in this field. The opinion of either one alone is of the highest value and when these three concur as a result of independent observations, as is the case in this instance, the weight of authority

is so overwhelming that contradiction seems futile; however, numerous arguments might be raised to the contrary. While it is not the intended purpose of this resumé to be controversial, yet as I understand the principles above outlined, there may be pointed out, for example, that if we accept Negus'[1] following explanation that "if air is expelled from the lungs with the glottis partially closed, the vocal cords are thrown into vibration; the air current is thereby divided into a series of puffs, and sound is produced" (the "reed" theory), it is necessary for us to assume that these vibratory movements of the cords are in the horizontal plane, a concept which is difficult to grasp. It would seem far more likely that these vibratory movements take place in the vertical plane, which, of course, is the direction of the powerful blast of air coming from below, or in several planes at the same time. Such a vibratory movement in the vertical plane would strongly support the "vibrating string" mechanism and mitigate against the "reed theory." The direction of the vibratory movement of the cords has not been definitely established. Certainly, stroboscopic observations cannot rule out a vibration in the vertical plane. The image conveyed to the eye through the stroboscope is a view from above looking down on the superior surface of the cords. Fine vibratory movements in the superoinferior direction, or vertical plane, cannot possibly be recognized by this method and a string-like vibratory movement has not, therefore, yet been ruled out. T. A. MacGibbon[24] supports this theory, although, as he points out, vibrations must take place in other planes as well. If the "reed theory" were to be accepted, it would follow, as Guthrie says, that alterations in pitch would not depend upon changes in the length of the cords. Yet it has been shown quite conclusively[3] that elevation in tone, especially in the higher registers, is accompanied by a functional foreshortening of the cords. Guthrie himself admits this in his discussion of the falsetto voice, and MacGibbon,[24] too, is strongly of this same opinion. This principle of functional foreshortening has been demonstrated even as early as 1886 by French, who presented a remarkable series of photographs before the American Laryngological Association, and since that time by others. Of course, not all pitch changes are due to shortening or lengthening of the cords — functional or anatomical — but when this does not occur, other factors such as an increase in tension or a narrowing of the chink of the glottis must be con-

sidered, and these phenomena, which certainly do take place, add nothing to the likelihood of a "reed" mechanism as opposed to a "vibratory string" mechanism. It cannot, therefore, be said that the question of a "reed" or "string" mechanism is definitely settled.—ED.]

Schatz[2] continues (but does not indicate whether it is his personal view or that of Russell) that "low tones are produced not by relaxed, elongated cords vibrating, but by a marked reduction in the size of the laryngeal outlet by approximation of the cartilages of Santorini and Wrisberg . . aided also by a loose approximation of the ventricular bands. This arrangement also serves to produce the guttural type of voice." [Towards this concept I feel free to take strong exception. In a discussion before the Royal Society of Medicine,[3] I demonstrated by motion pictures that the very low guttural tone is actually produced by markedly relaxed vocal cords, which are not elongated but foreshortened, and that there is present, not as Schatz indicated, a reduction in the size of the laryngeal outlet but, on the contrary, it would appear to be comparatively enlarged. I agree, however, that there is present a loose approximation of the ventricular bands. Schatz in the above discussion uses the phrase, "relaxed, elongated cords." To me, this is paradoxical, insomuch as it can easily be demonstrated that when a vocal cord is relaxed, it is not elongated, but rather foreshortened. "Relaxation" and "elongation" of vocal cords cannot ordinarily take place simultaneously.—ED.]

Negus[1] and Schatz[2] in their respective publications discuss more than just the "reed theory" of sound production. Negus, for example, includes a brilliant discussion of "resonators." He points out that sound produced in the larynx is but a feeble and colorless effort requiring the superposition of various resonators. He stresses the fallacy of dividing the human voice into "head tones" and "chest tones," on the assumption that they are chiefly associated with resonance in one or other of these regions. Negus points out, to the contrary, that all the resonating cavities connected with the airway are actually in action during all tones. Thus the trachea and bronchi play their part as resonators. The oropharynx in man is likewise an effective resonator, which is genetically accidental since its primary function is its rôle in swallowing. The nose and naso-

pharynx are available as resonators because the mechanism of nasopharyngeal closure during swallowing has been modified for purposes of phonation. The relatively large nasopharynx and small oral cavity of man as compared with lower animals is of great advantage, making it possible for "double resonance" to be effected. This is important for variations in the quality of sound.

In these resonating chambers, overtones which, like the fundamentals, find their origin in the larynx, are modified by being strengthened or suppressed. This modification of overtones likewise occurs in the larynx itself.

[It would be difficult to imagine a more scholarly or interesting treatise on the subject of the physiology and evolution of the larynx than Negus has prepared. I recommend, most enthusiastically, that it be carefully studied by all students of the subject.—ED.]

Concerning this question of resonance, Russell[14] has contributed a most interesting study to supplement Negus' observations. He and a number of members of the Acoustical Society observed a patient in whom "all the sinus and other open cavities above the velum and palate had been completely removed. There remained nothing but one wide, megaphone-like opening in the head, where they and the nose should have been." It was agreed that the carrying power of this individual's voice was not impaired but actually was greater than normal. Russell interprets this as indicating that resonance in the voice cannot, therefore, be traceable to any sinus functioning as a resonator, "for every paranasal sinus in this man's head was gone." [I, for my part, cannot, however, accept this as proof positive, since Russell has failed to take into account that the "wide, megaphone-like opening in the head" may have provided an even greater resonating chamber than the nasal accessory sinuses it replaced.—ED.] However, Russell quotes further more convincing experiments in support of his theory that the nasal accessory sinuses play no rôle in the resonance of the human voice. He describes experiments carried out with Warren at the University of Wisconsin, in which an artificial larynx was sealed into a cadaver head and an oscillographic tracing made of sounds produced after filling the various sinuses, and varying the size of the ostia. The results of these curves showed no deviation from

the normal in loudness or quality, regardless of whether the sinuses were filled or empty. [Such evidence is important and calls for considerable modification of pre-existing ideas of the nasal sinuses as "resonators." It has, too, great clinical value especially as it relates to indications for sinus surgery in vocalists.—ED.]

There can be no doubt, however, from repeated observations in pathological states where filling of the sinuses occurs, that voice changes are an invariable sequelae. Russell[15] suggests that the source of such changes in voice be sought in the accompanying congestion of the turbinates and muffling effects due to inflammation elsewhere. Russell[17] has also proven that an opening of a resonator no larger than those leading into the sinuses is so small that no sound is radiated by it loud enough to be heard across the room.

Guthrie[13] lends considerable support to Russell's theories. He points out that the ostia are small and hidden behind the middle turbinal away from the main air current. He reminds us that in song or speech, the air does not pass out by the nose save in the sounds of m, n and ng, and cites the fact that there is no proof that operative destruction of the walls of the sinuses has any effect on voice. He believes the chief resonators to be the mouth and pharynx, omitting mention of Negus'[1] description of resonators below the larynx.

MacGibbon,[24] in a personal communication discussing Guthrie's[13] paper, takes strong objection to the opinion that the sinuses play only a small rôle, if any, in vocal resonance. He points out that no ostium at all is necessary since sounds pass readily through solids, and feels strongly that the sinuses add to the vibrations of the air columns above the cords, and that these give an added richness to the quality of the high tones of the human voice. [Despite the weight of evidence so brilliantly brought forth by Russell and Guthrie, it would seem that MacGibbon's reasoning is sound, and his conclusions not without some basis of fact.—ED.]

Schatz[2] emphasize that good singers give evidence of exceptional voice before coming under the guidance of professional voice trainers, and their voices are not the product of any personal method of teaching. The anatomical parts which co-operate in producing a voice of a particular power and quality are the trachea, size and structure of the laryngeal

cartilages, the extrinsic and intrinsic muscles of the larynx, the pharynx faucial space and oral cavity. He omits, whether accidentally or by intent, any mention of the nose and nasal accessory sinuses. This author stresses the rôle of the epiglottis in modifying and directing the column of air, and points out the important rôle played by the character of the muscular supraglottic tube and the expansion it can undergo. He also stresses the need for proper functioning of the mucous glands if pleasant sounds are to be produced. Schatz draws a finer distinction between "chest" tones, "head" tones and "intermediary" tones than does Negus or Guthrie, who believe these registers are not at all anatomical but rather represent the site in which the vocalist feels the tone, which does not mean that they are formed in these parts. Schatz describes the various positions in the neck assumed by the larynx in producing these registers. The larynx ascends in position and the glottis becomes more horizontal as the scale ascends. Guthrie,[13] however, limits the elevation of the larynx as the scale ascends to the untrained singer. In the trained singer, this movement appears to be reversed and the larynx may actually descend as the pitch rises.

Curry[18] stands with Schatz, and as a result of his Roentgenographic studies has concluded that for the highest pitches the larynx is drawn up closer to the hyoid bone. This progressive upward movement of the larynx must involve increasing tension in the internal laryngeal muscles, including the vocal cords. This increasing tension may result in the narrowing of the larynx ventricle vertically, and in a closer approximation of the ventricular bands to the vocal cords. Thus, the mechanism of pitch change in the voice involves not merely tension of the vocal cords but also bodily upward movement of the whole larynx.

Schatz continues by suggesting that in the higher tones the epiglottis, too, is elevated out of the way of ascending air currents, permitting them to reach the upper respiratory tract without obstruction, producing "head tones," whereas in the lower tonal range or "chest tones," the opposite is the case. In vowels such as the "e" in "peep," the pharyngeal pouch is enlarged to its maximum, and the epiglottis farthest removed from the posterior surface of the larynx. The pharynx is reduced to its smallest diameter in "a," as in "pap." Schatz

points out that the soft palate is raised in most singers and yet does not interfere with proper head resonance, and stresses the importance, if proper laryngeal vibration is to take place, of relaxation of those extrinsic muscles which hold the larynx in its position in the neck. The author quotes MacEwen as opposing enucleation of tonsils with their capsules because of damage to the stylopharyngeus. Any injury which cicatrizes the pillars of the fauces [presumably including tonsillectomy.—ED.] can also hamper the voice. [There are innumerable examples to refute this. Occasionally a short period of readjustment is necessary, but I have not yet seen permanent damage to any voice as the result of properly performed tonsillectomies.—ED.] Schatz then continues with a number of practical suggestions for voice training, all of which seem commendable. The rôle sinusitis plays in affecting voice is believed to be only an indirect one, and due to drying of the pharyngeal mucosa, or by causing spasm in the neck muscles, or because of a secondary cervical adenitis. Pathologic changes within the sinus mucosa, however, have no deleterious effect upon head resonance. General bodily fatigue affects muscle tone and function, thereby impairing good tone production.

Pressman[3] reports certain laryngeal phenomena which he has been able to observe in motion pictures taken of human subjects under local anesthesia. His observations include laryngeal movements during respiration, phonation and swallowing. The area of the chink of the glottis adequate for respiratory needs during physical inactivity does not permit the passage of sufficient quantities of air during physical exercise or excessive vocalization, as in singing. Under these circumstances, the glottic chink is widened by abduction of the cords, the degree of abduction possible being increased by proper respiratory training. In especially well trained singers we find the cords can be abducted to an extreme degree, offering almost no obstruction to the rapid inhalation of large quantities of air.

Canuyt, Gunsett and Greiner,[20] whose paper is discussed more fully in another section of this resumé, report by Roentgenographic studies similar conclusions concerning this extreme abduction of the cords during the inspiratory cycle. Kistler[23] points out that this abduction is an involuntary act,

under the control of the respiratory centre in the medulla rather than being under the control of the higher cortical centres.

He (Pressman) mentions the speed at which vocal cords can undergo cycles of abduction and adduction, these being very rapid, and under certain conditions exceeding 16 cycles a second. [Further observations would tend to indicate that it is not possible to accurately estimate numerically this potential speed of movement, but it can safely be said that the cords can undergo cycles of ab- and adduction more rapidly than the eye can follow them.—ED.]

There is a discussion of the function of the false cords which are not remnants of vestigial structures but, on the contrary, play a definite rôle in laryngeal physiology. During the swallowing act, they help to protect the lower air passages from the invasion of foodstuffs by tightly approximating in the midline and "sealing" the larynx. Under certain pathological circumstances, they tend to take over the function of the true cords and act as phonatory organs. These observations are, in part, supported by Russell.[14]

In describing the appearance of the cords during phonation, the author tends to disagree with the "reed" theory supported by Negus, Guthrie, Russell and Schact, and speaks of the cords as "vibrating strings." He is able to demonstrate that the visible vibratory movements of the cords take place only in the anterior two-thirds, suggesting that this may have something to do with the site of vocal nodes.

Three mechanisms are demonstrated by means of which tones of varying pitch are produced. Any given tone is believed to represent a combination of all three demonstrable principles of sound production. The first is a tightening or increase of tension as the scale ascends. The second is that of "functional (not anatomical) foreshortening." This is accomplished by the more posterior portions of one cord coming into contact with and damping a corresponding length of the other.

T. A. MacGibbon,[24] agrees that such damping occurs, and explains it by the fact that the internal thyroarytenoids which arise at the vocal process of the arytenoid cartilages contain fibres which terminate in the vocal cords themselves. These

bands act like the fingers of a violinist and "damp" or "stop" the cords, thus raising the pitch. [This may be one explanation of the damping process, but it cannot be denied that such damping also takes place by the contact of portions of one cord against the other. More than likely, MacGibbon's theory may be the anatomical mechanism by which selective contact of segments of the cord is made possible.—ED.] That such selective action of certain fibres of the thyroarytenoidii can take place is supported by Kistler,[23] who indicates that man can control not only the movements of the laryngeal muscles themselves but almost of individual fibres or groups of fibres, and the more highly developed is this control in one individual as compared with another, the more effective becomes the speaking and singing voice. Thus, by this damping a shorter segment of each cord is free to vibrate and a higher tone produced. As higher and higher tones are produced, the length of cord in approximation with its fellow increases and the vibrating segment becomes shorter and shorter. The third principle by which higher tones are produced is the natural result of the second. As greater lengths of the cords come into contact with its fellow, the chink between them becomes smaller and, as can be observed in whistles, for example, the smaller the lumen through which air passes the higher pitched will be the sound produced. The elevation of pitch of a tone results from a combination, then, of increased tension of the cords, shortening of the vibrating length by damping of segments of one cord against the other, and a narrowing of the glottic chink. All of these phenomena are demonstrable upon the motion picture film. [These observations, while undoubtedly accurate, do not in themselves make untenable the "reed theory," since the two are not entirely contradictory.—ED.]

Newhart[4] discusses the importance of the relationship of deafness to speech defects. He points out that since speech is acquired chiefly through imitation by hearing, that subnormal hearing must cause a very substantial proportion of speech defects. To substantiate this, he quotes Russell, who points out that speech defects are eight times more frequent in the hard-of-hearing than in the normal. Even a slight hearing loss may cause certain speech defects. Thus, a child having a relatively small hearing loss in early life often suffers greater speech distortion than a child having a much greater loss later in life. Newhart suggests that, contrary to popular belief,

even stuttering may in a casual way be due to hearing loss because of resultant anxiety, frustration and fatigue incident to the prolonged effort to hear. The most important factor in the relationship of imperfect hearing to speech defects is the early recognition and, when possible, the early correction of the deafness.

Remnants of hearing, when supplemented by hearing aids, lip-reading and visual hearing, are priceless aids in learning and retaining correct speech.

Max A. Goldstein[11] similarly stresses the relationship of speech defects to defective hearing. This relationship exists because speech is only an imitation of sounds we hear, and if heard imperfectly cannot be properly imitated. Even when defective hearing is amplified by hearing aids, the model of speech heard can never be perfectly interpreted and reproduced. Vision and sense of touch are our approach to teaching speech to the deafened. This approach is an arduous task, requiring a world of patience and understanding of the speech mechanism, the hearing organs, the value and application of tactile impression, and an understanding of the psychological urge and scholastic ambitions of the child. Added to this knowledge is the aid possible from audiometers, the oscillograph, the strobilion and the phonelloscope. With these aids, an experienced teacher can teach the deaf fluent speech with flexibility, pitch, accent, rhythm and volume control.

The other approach to the totally deafened is lip-reading.

Goldstein can point with satisfaction [and pride—ED.] to the well trained totally deaf pupil, age 4 to 7 years, who in later life can compete scholastically and economically in the speaking world with his normal hearing fellows.

In closing, Goldstein urges close co-operation between the otologist, teachers of deaf and the speech correctionist.

Tilley[5] discusses a series of cases of hoarseness due to an inactive but otherwise normal vocal cord, many of them not traceable to a lesion of the corresponding recurrent laryngeal nerve. The first of these represents permanent ankylosis of the cricoarytenoid joint following a severe bacterial laryngitis. The second and third are cases of severe acute laryngitis with edema, in which a temporary fixation of the left cord occurred. Then follows 14 cases of "chronic hoarseness"

due to immobility of a vocal cord, the involved side being the left, except in a single instance. Most of these cases were due to previous inflammatory lesions involving the larynx, or to blood-borne bacterial toxins or chemicals. The author points out the far greater vulnerability of the left cord even in those cases where the longer course of the left recurrent laryngeal nerve plays no rôle. Practically all these cases ultimately recovered normal function.

There is then pointed out that hoarseness may be due to a laryngeal nerve neuritis dependent upon avitaminosis, especially beriberi. No explanation is given for the overwhelming disproportion between right and left cord fixation in those cases due to local inflammatory reactions or circulating toxins. In contrast to this, Orton[7] describes in cancer of the larynx a greater frequency of immobility of the right cord as compared with the left. [Except for recurrent laryngeal nerve lesions, which for well known anatomical reasons are more apt to occur on the left, there seems to be no reason for this peculiar distribution, and it is questionable whether such a small series is of significance.—ED.]

Leroy Schall[6] indirectly points out the rôle played by the larynx as an essential organ for appreciation of the odor and flavor of foodstuffs. Following laryngectomy, the inability to inhale through the nose greatly impairs these functions. The larynx, too, is an organ essential to proper swallowing since after laryngectomy food tends to become lodged in the throat and can be dislodged only by swallowing liquids.

Negus[8] describes changes in the cricopharyngeus muscle following laryngectomy, which changes tend to promote the development of an esophageal voice. The new sphincter formed after suture of the pharynx has less control over the entrance of air than in the normal individual and so air-swallowing becomes easy. The faculty for drawing air into the esophagus and stomach is, therefore, easily learned. It appears probable that the constriction formed by the sphincter may even act as a reed and thus be the origination of sound for use in speech.

Taft[9] stresses the value of oscillographic tracings in the study of vocal characteristics and describes a simple method for making a satisfactory oscillograph at nominal cost.

Eisenson and Winslow[10] describe the phenomenon of "perseveration" as playing an important part in stuttering. The perseverating tendency is one in which ideas, once they have occurred, remount into consciousness spontaneously. The production of a speech sound results from excitation of sets of neurons which, once excited, tend to persist in the original state of excitement and show resistance to any change in this state. This, in stutterers, manifests itself by the repetition of a sound once it has been uttered. Stuttering represents, then, the same general neurologic phenomenon as the sensation of "sea legs" after a sailor has come ashore.

Paul Moore[12] describes the results of observations of laryngeal physiology made from slow motion moving pictures. He draws only two conclusions: *1.* That the laryngeal movements preparatory to vocalization are very complex; and *2.* that the so-called "stroke attack" involves more constriction in the superior laryngeal musculature than the "breathed" or the "simultaneous" attacks. He does not, however, define these terms. The author points out that slow motion pictures at 120 frames per second do not present the individual vibrations of the vocal folds. Ultra high speed photographs taken in conjunction with the use of a stroboscope demonstrate that "the vocal lips [*i.e.*, vocal bands or vocal cords—ED.] perform a complete wave-like vibratory movement. The wave starts with an opening between the anterior sections of the lips and progresses posteriorly as the folds separate. The closing phase of the movement appears to travel anteriorly from the arytenoid region. In such vibration the glottis remains open longer anteriorly."

Douglas Guthrie[13] introduces a most comprehensive and stimulating article by pointing out the lack of attention paid to matters pertaining to voice production, and the difficulty of finding synopses of the present day knowledge of the mechanics of voice, especially in modern standard texts on laryngology. This, he believes, is due to the very many sciences necessarily involved in the study of voice. He describes the conflict existing between singers and scientists, as exemplified even in the use of a terminology by one group, which terminology is not understood by the other. The vocal cords are capable of delicate adjustment by the action of numerous muscles, some 60 in number. Concerning the devel-

opment of the voice, it is to be noted the newborn cries on a note approximating 435 cycles per second, the vocal range extending to six and a half tones and reaching an octave at the end of four years. Just before puberty, this stretches to an octave and a half, which limited range should be taken into consideration by those who compose children songs, which are often ill adapted to their voices. [This, and other limitations of the youthful larynx, should likewise be considered in the training of young voices, which anatomically are ill fitted to undergo the rigors of professional usage. Many promising youthful vocalists suffer ruination of possible future careers by too early and too vigorous training.—ED.]

West[16] is of the same opinion, bringing out the point that irregularities in the opening between the cords in adult life, causing imperfections in the voice, may be traceable to too vigorous use of the voice long before the larynx has developed a sufficiently strong and rigid framework to resist the exaggerated pull of the extrinsic and intrinsic muscles. Under these circumstances, the larynx becomes "warped," leading to irregularities at the opening of the chink of the glottis. Usually this manifests itself by a too wide opening between the arytenoids furnishing such a ready egress to the air stream that not sufficient pressure is exerted on the membranous cords. Again, it may result in abnormalities in the position of the epiglottis. Expired air striking against the backwardly tilted epiglottis causes a back pressure upon the vibrating cords, stopping these vibrations and distorting the sound.

[This, I think, is a new conception of the mechanism of voice injury from overuse in early childhood. We are, ordinarily, accustomed to look upon the etiology as a traumatic inflammatory laryngitis causing thickening, erosions, or exaggerated vocal nodes. West does not deny that this, too, occurs.—ED.]

To continue with Guthrie's[13] article, he reminds us that at puberty the male larynx grows rapidly and undergoes "breaking," which may begin as early as age 8 years or as late as 18, the transition taking from six months to two years. In boys, the vocal pitch falls by one-sixth at its upper limit and by an octave at its lower limit. Hoarseness is common and in about 10 per cent of the cases there may be seen a slight

redness of the vocal cords and laryngeal mucosa. In girls, the change is much less pronounced, more rapidly completed, and consists merely in a slight extension of the upper and lower limits. Incomplete changes result in a persistent falsetto voice, and perverse changes take place which account for the occasional appearance of the female bass or the male soprano. In boys who have undergone early castration no vocal changes occur. The ordinary adult human voice has a range of two and one-half octaves, seldom extending over three. The entire compass of the human voice, from the lowest bass to the highest soprano, is six octaves. In ordinary speech, a range of about an octave is used, and in dramatic speech about two octaves. In men, the average speech range is situated at about 140 cycles per second, and in women at about 243 cycles.

Guthrie points out that the vocal mechanism consists of three parts: respiration, phonation and articulation, the last being intimately associated with resonance. He describes the various respiratory mechanisms which may be used in production of the singing voice, including the technique of "rib reserve," which had not previously been described. Incorrect breathing is perhaps the chief cause of vocal failure.

In a plea for co-operation between the teacher of speech and the laryngologist, West[16] stresses the importance of an adequate laryngeal examination prior to the commencement of a regime of speech training. This is not only to rule out morbid states, but to study the larynx as a producer of voice. The characteristics of the ideal glottis are these: *1.* It must have two edges that can be brought into exactly parallel approximation. *2.* This approximation must be so close as to prevent the escape of any air except when driven apart by a real chest pressure. *3.* The approximation must not be so close as to prevent the free movement of the bands when set in vibration by the air stream. Apart from this, West discusses certain anatomical deficiencies, such as a broad gap between the arytenoids during phonation, causing a voice "aspirate" in quality. In the ideal larynx, the arytenoid glottis is a continuation of the same straight line as the membranous cords.

Flurin[19] discusses at some length the effect of the endocrine system upon laryngeal physiology. The function of the larynx is closely related to the endocrines, predominantly the sexual

glands, which, however, must be considered only in relationship to the other endocrines. Terracol and Azemar are widely quoted and much of what follows is their work.

Endocrines play a rôle in the development and function of the larynx and in the anatomy and pathology. The development coincides with the maturation of the sex glands. The cartilages are chiefly involved, rapidly calcifying due to the function of the parathyroid in controlling the deposit of calcium. The close relationship of the larynx and sex glands brings about a change in the female larynx at the menopause.

In capons (intermediary sex character), the larynx degenerated. Roosters crowing could be prevented by castration. The injection of posterior lobe of hypophis extract affects the crowing of roosters, but the author does not say how.

In man, castration results as follows: *1.* Before puberty, the development of the larynx is prevented. *2.* After puberty, castration does not abnormally modify the larynx, except that calcification is slower in developing and starts later, approaching more closely the development of the feminine larynx. The voice of the castrate does not have either a true masculine or feminine quality, but is typical only of itself, resembling more closely that of boys.

Other endocrines in castrates cannot replace function of the testicular extract in affecting the tonicity of the musculature of the larynx. The testicles control the development of the physiological mechanism of the larynx.

A eunochoid type of voice may develop from extragenital causes, and if the larynx is normal anatomically this can usually be corrected by voice training.

In the female, castration may bring about a lowering and roughening of the tone, but masculinization is unusual in the normal menopause.

The male hormone must be present if the changes in the larynx at puberty are to occur.

The sexual characteristics of the voice (male and female) remain during the sexual life of the individual. Sexual continence in singers maintains them in better voice. The other endocrines besides the sex glands play a rôle in laryngeal

development and physiology. Laryngeal congestion can occur from genital origin.

During menstruation, vocal changes are frequent and the voice fogged. This menstrual dysphonia is due to a hyperemia, chiefly of the vocal bands. These changes are most marked just before menses.

Pregnancy rarely affects the voice, but whether it is endocrine, mechanical or gravid autointoxication is not known.

There is a definite vocal change at menopause — the voice becoming masculinized. The limits of the high register diminish, and the lower limits increase, but a beautiful voice may still remain beautiful after menopausal narrowing of range. Exercising the voice tends to prevent the development of those changes due to age. These changes at menopause may be very variable. Various types of pelvic pathology (tumors, inflammatory reactions, etc.) causing other secondary sex changes are accompanied by similar changes in the larynx.

The thyroid gland plays a frequent rôle in causing obscure laryngeal difficulties. A "dysthyroidism" may account for hoarseness without demonstrable local causes. This is a transient but recurrent type of hoarseness, accompanied by difficulty in producing all sounds and accompanied by a lessening of good timbre. This "thyrovocal" syndrome occurs apart from other hypo- or hyperthyroid symptoms. This is supposed to be related to close inter-relationship of the blood supply of the thyroid gland and the larynx. This syndrome is, then, mechanical and due to vasomotor changes rather than due to the abnormal thyroid secretion which gives the other symptoms of hypo- or hyperthyroidism.

In Basedow's disease, the vocal changes are variable, mostly a sensation of fatigue. The pitch is often elevated. The notes are trembling, not true and not well sustained, sharpness of sound is not so acute, and the timbre "raucous." In speaking, the patient may suddenly lose his voice, which may return after swallowing. This is an emotional state comparable to stuttering.

In suprarenal hypersecretion we find a "commanding" voice in males and a contralto voice in females, along with the other attributes of virulism. In hyposuprarenalism we find the

reverse. In Addison's disease, vocal changes occur later, and vocal fatigue is more marked than in Basedow's disease. It progresses as the disease progresses to the point of total aphonia. These syndromes are often closely inter-related. The vocal changes in these hyperthyrosuprarenal pathological states are due to local vasoconstriction and hypertension secondary to hypersympatheticotonia. The hyperendocrine states lead to an exaggerated development of the larynx, and in the hypo- states, this development may be greatly retarded.

Canuyt, Gunsett and Greiner[20] have studied the appearance of the larynx under varying circumstances by a procedure known as "tomography." X-rays are taken in the antero-posterior plane in such a manner that clear front views of the larynx are obtained without the superposition of the cervical spine. The Roentgenograms demonstrate that in inspiration and expiration alike, the vocal cords, ventricles and false cords are completely effaced, blending one into the other along the lateral wall of the larynx. [This is obviously a distorted image, since by direct observation and photographs, it can be clearly shown that only upon very deep inspiration, and then only in a few certain selected individuals, does this blending into the lateral wall occur. It certainly is not true of expiration under any circumstances; therefore, it is quite impossible to attach much weight to the remainder of the observations described as having been made by this method.—ED.] In apnea, the two laryngeal walls come together *en masse* in the midline. [This is certainly true of "spastic apnea" but equally untrue of other apneic states, especially of central origin, in which the cords usually occupy a more or less "cadaveric" position.—ED.] The authors describe the Roentgen appearance of the larynx during the production of vowel tones. For example, in producing vowel "o" the ventricle opens more widely than in producing "e," and producing "i," the ventricular bands are separated from the vocal cords by a very large space, more so, for example, than when producing "a." In the former case the anatomic formations are very clear. During the production of higher pitches, the ventricular bands are thicker, larger, more voluminous and nearer to each other than in the lower voice. [This is, I think, contrary to most observations.—ED.] The true vocal cords are thinner and more flattened and more closely approximated one to the other. The vertical length is increased. In low "chest" tones

the ventricle becomes almost completely obliterated, due to approximation of the vocal bands to their respective vocal cords, and these structures show a more rounded or globular form than during the production of high tones. The vocal cords present a progressive narrowing during the production of higher tones, and the space between them becomes less.

Changes in the timbre and quality of the voice found in epidemic encephalitis, Parkinson's disease and pseudobulbar palsy is the subject matter of an article by Schiff and Labarraque.[22] They describe an overlapping and failure of elevation of the arytenoids observed in a number of these patients. The peculiar qualities of the voice are due to abnormal tension of the vocal cords, with partial or generalized tremblings resulting from changes in muscle tonicity. In Parkinson's disease, a hypertonicity is present, while in encephalitis the reverse is true. [I have observed and photographed such overlapping of the arytenoids as the authors describe, in a patient who may well have an early central nervous lesion, the exact pathology of which cannot yet, however, be accurately diagnosed.—ED.]

Laryngeal physiology as affected by unilateral recurrent laryngeal nerve paralysis has been clinically studied and described by Kistler,[23] although he adds very little that is new. We see during spontaneous or therapeutic healing of recurrent paralysis that during phonation the paralyzed cord is relaxed, thinner and remains immobile in an intermediate position. On the other hand, the healthy cord makes an exaggerated compensatory hyper- adduction, with or without crossing over of the arytenoid cartilages. By these means, approximation of the cords and resultant phonation is made possible. This compensation of movement takes place because of the close association in cortical centres between the nerve fibres of the two sides. This bilateral symmetrical cortical control insures one side functioning more effectively when the other functions less effectively. The author points out that control of abduction of the cords is dominated by the respiratory centre in the medulla and is more or less independent of higher centres. On the other hand, the phonatory adduction apparatus is controlled by the cerebral cortex, but it is not known whether there is a definitely specialized cortical centre. The author points out the ability of man to control the movements not only of the laryngeal muscles themselves but almost of individual fibres or groups of fibres. The greater is this control

in any individual, the more effective becomes his speaking and singing voice. Through practice, this conscious control can be increased very appreciably. This effective control mechanism enables the larynx to functionally compensate in a similar way for pathologic changes that may be present, and eventually to develop a satisfactory mechanism different from the normal, but functionally effective despite the pathology. Very often we find individuals who develop only partial healing and compensation. As a result, there develops a false vocal cord voice. These are usually individuals who did not have good control of the larynx even when normal. [A most interesting case under my observation which illustrates this point is a man with extensive bilateral polyposis, who for many years phonated exclusively with his false vocal cords. Following appropriate surgery and the re-establishment of a satisfactory anatomical appearance to the true cords, he continued, as demonstrated by motion pictures, to phonate with the false cords. The true cords, although now anatomically sound, still continue to be physiologically without function.—ED.]

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1917 Wilshire Boulevard.

OPERATIVE PROCEDURE FOR THE RELIEF OF STENOSIS IN DOUBLE ABDUCTOR PARALYSIS OF THE LARYNX.*

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The treatment for stenosis in bilateral abductor paralysis of the larynx resolves itself into two fundamental procedures: *1.* Emergency treatment, when necessary, to save the life of the patient. *2.* Treatment directed toward producing a permanent and adequate airway through the natural air channels.

The emergency measure is immediate tracheotomy to relieve dyspnea. MacKenty's[1] operation may be useful as an intermediate step in order to do away with the tracheotomy tube during the time the recurrent laryngeal nerves or nerve centres may be recovering from damage or disease.

In producing a permanent airway, some physiological procedure which will preserve the voice should be the method of choice.

Medical treatment should be directed toward any systemic disease causing the paralysis, and sufficient time should be allowed for functional recovery of the larynx before any type of surgery is considered.

If the recurrent laryngeal nerves have been cut accidentally, suturing the cut ends should, naturally, be the logical treatment. Unfortunately, this operation has had little success up to the present time. It is hoped that the technique of small nerve suturing will advance in the future to the point where this procedure will be more satisfactory. Recurrent laryngeal nerves have such a long and tortuous course that, apparently, it has not been necessary to attempt nerve grafting. Anas-

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tamosing the recurrent laryngeal nerves with other nerves has not been very successful.

Paralysis due to pressure from without or to scar tissue contraction following thyroid or other neck operations necessitates eliminating the tumor causing the pressure or releasing the nerves from the scar tissue.

Cutting the recurrent laryngeal nerves in order to produce a cadaveric position of the cords has met with no success. The possibility that there is sometimes an anatomical variation in the innervation of the arytenoid muscle may make a difference in the results of some types of treatment. Lemere[2]

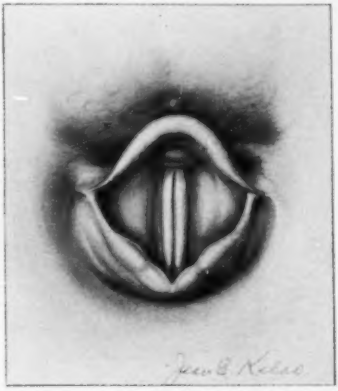

Fig. 1. Appearance of larynx before operation.

has demonstrated that blocking the external rami of the superior laryngeal nerves will, in some cases, give considerable relief from the accompanying dyspnea. He attributes the relief to the relaxation of the nonparalyzed cricothyroid muscles. Imperatori[3] believes that when the superior laryngeal nerves innervate the arytenoid muscle, the relief will be greater.

In paralyses due to central nervous system pathology or to neuritis from diphtheria, influenza,[4] lead poisoning, etc. or in any case where atrophy and fibrosis of the posterior cricoarytenoid muscles has developed, some nonphysiological surgical treatment[12] may be necessary. It is with this type of case that we are concerned in this paper.

Plastic operations to widen the anterior portion of the larynx have met with little success. Surgical removal of the cords and external transplantation[5] have proved to be of little merit. Ventriculectomy did not work out on humans.* Simple removal of the arytenoid cartilages alone was not sufficient. Ventriculocordectomy[6] has been fairly successful in the Jackson Clinic, but has not given satisfactory results in the hands of others.

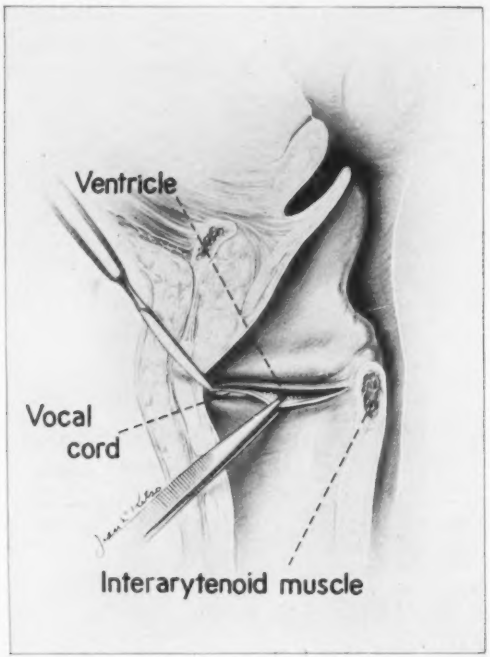

Fig. 2. Incision is made above the cord and the mucous membrane elevated over and below the cord.

In 1934, Rethi,[7] a Hungarian, described a new and successful operation relieving stenosis in bilateral abductor paralysis. After doing a laryngofissure and elevating the mucous membrane over one side of the larynx from the cord and arytenoid downward, he severed the adductor muscles from the arytenoid cartilage. He then cut out a strip of

*Ventriculectomy was very successful on the horse.

mucous membrane below the incision and sutured the margins across the gap to hold the cord and arytenoid in a more external position. The laryngofissure was kept open with tampons for 15 days, and the mucosal sutures were removed on the seventh day. It was necessary to do this operation on only one side. Of the 16 patients operated, all but one had sufficient airway. All developed poor voices following operation.

Rethi[8] had previously (1927) reported a somewhat similar operation in which he divided the adductor muscles, and then displaced the cord and arytenoid externally and held them in position with tampons until an ankylosis of the cricoarytenoid joint was produced. He cited two cases operated successfully in this manner.

Rethi's results were excellent, but his procedures were somewhat complicated and the postoperative care required a considerable period of time. However, his work merits serious consideration.

Hoover's method of submucous resection[9] of the cord along with removal of the vocal process of the arytenoid was fairly successful and a definite step in the right direction. In this operation, it was sometimes necessary to do a bilateral resection in order to produce sufficient airway.

Loré had worked out this technique independently when Hoover reported his work. While Loré believed that it was the most promising operation of that time, he did not believe that it achieved all the desired results. He, therefore, continued his studies and in November, 1935, gave a preliminary report,[10] and in September, 1936, presented a complete anatomical study of a new operation which had been worked out on cadavers.[11] He stated frankly that he did not know what the end-results would be on a living subject.

Loré believed that other similar operations had failed because the new, adventitious cord always came back to the same midline position that it had previously held. The manner in which this occurs he described as follows:

"It must be assumed that the vocal cord has two points of attachment: One is the anterior commissure and the other is the vocal process of the arytenoid. It is the constant pull between these points which limits the cord to the midline."

Therefore, he decided that shifting one attachment of the cord to a more external position and at the same time removing the pull of the thyroarytenoid and lateral cricoarytenoid muscles would accomplish the desired result. Working on this theory, he developed an operation on cadavers in which he did two fundamental things: *1.* Removed the adductor mus-

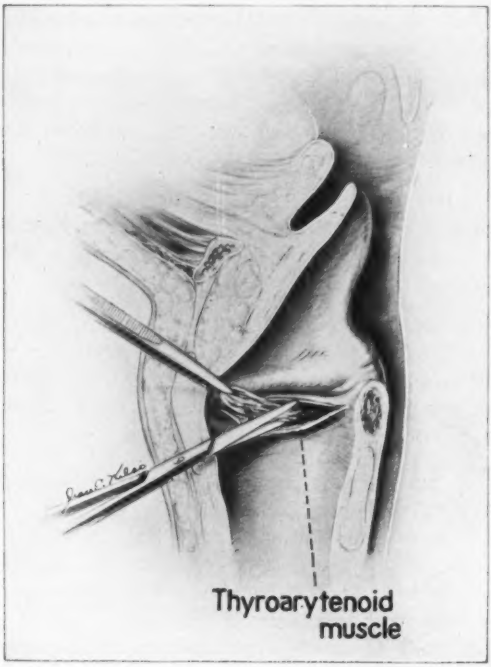

Fig. 3. Showing removal of tissue of the cord between the mucous membrane and the perichondrium (mostly thyro-arytenoid and lateral cricoarytenoid muscle).

cles that make up the cord. *2.* Removed the entire arytenoid cartilage.

Believing that Loré's reasoning was sound and logical, and that his work offered the best possibilities for a good result, I did this operation on a patient with bilateral abductor paralysis and following is a report of the case:

History and Findings: Mrs. A. L. B., now 63 years old,

at the age of 24 years, developed a very severe sore throat which was probably diphtheria. Following this she was very hoarse for about a year and a half. Her voice gradually returned to normal, but, concurrently, difficulty in breathing developed.

At the age of 38 years, she had a tonsillectomy and almost died while under the anesthetic. She then had an exploratory

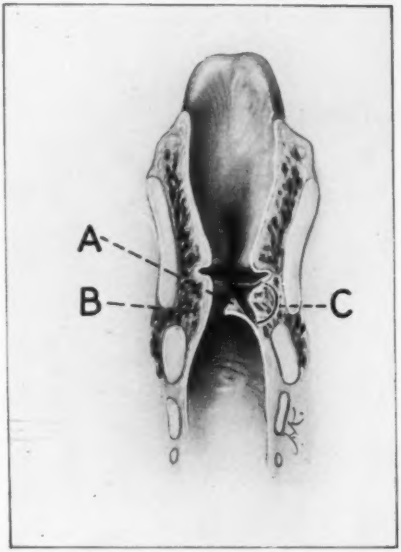

Fig. 4. A. Elevated mucous membrane. B. Muscles of the cord. C. Depth to which tissue is removed.

operation for a possible substernal goiter, but nothing unusual was found.

Six years later, marked dyspnea developed to the point where she had marked crowing and stridor on inspiration. There was less difficulty on expiration. The dyspnea gradually became so severe that the patient almost died during an acute upper respiratory infection, and it was following this episode that she first consulted me for relief.

On examination, the general physical and neurological findings were negative, as were the blood Wasserman and spinal

fluid Wasserman tests. X-rays of the neck and mediastinum showed no pathology.

The larynx on examination revealed both cords in the median position and on the same level (see Fig. 1). There was a slight retraction on inspiration and very slight fluttering opening on expiration. Her functional voice was rela-

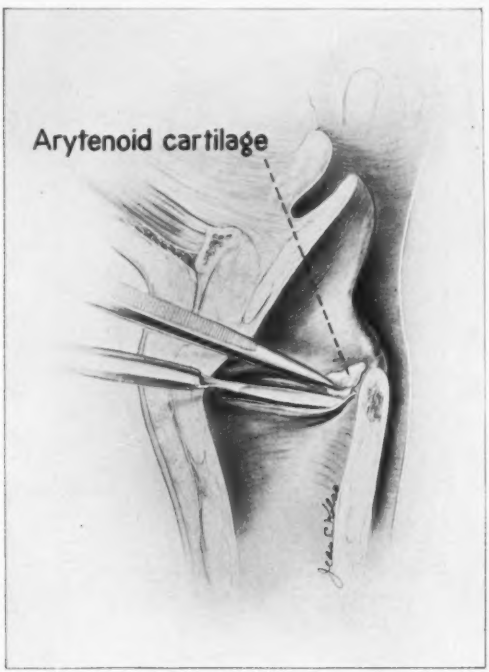

Fig. 5. Removal of arytenoid cartilage.

tively good, but she had to stop every few words to gasp for air. There was no fixation of the cricoarytenoid joints. The diagnosis was bilateral abductor paralysis, possibly due to diphtheria.

Operative Procedure: The anesthetic used was avertin rectally with novocain and adrenalin locally. The superior laryngeal nerves were blocked, and novocain was injected over the thyroid cartilage and trachea. Preliminary tracheotomy was

done, and the larynx well exposed by means of a laryngofissure. To prevent secretion from going into the lower respiratory tract, the trachea was packed off with gauze. The laryngeal mucosa was packed for five minutes with equal parts of 20 per cent cocain and adrenalin.

From this point, I varied my procedure somewhat from that described by Dr. Loré in his anatomical study.

Making a horizontal incision just above the left cord, from the anterior to the posterior commissure, I elevated the

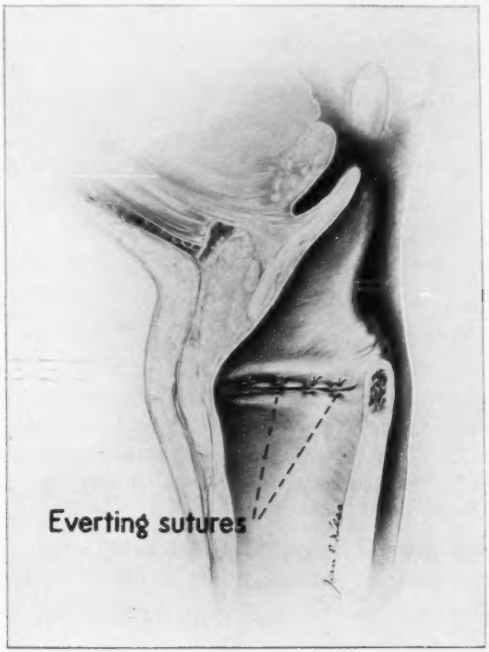

Fig. 6. Closure of incision. A small right-angle incision at the posterior commissure can be made if necessary.

mucous membrane over and below the cord on the left side (see Fig. 2.)* Starting at the anterior end and working back to the arytenoid cartilage, the thyroarytenoid muscle and other tissues making up the cord were removed down to the perichondrium of the thyroid cartilage (see Figs. 3 and 4). The

*Drawings show operation done on right side.

arytenoid cartilage was then dissected free with a blunt dissector and a pair of small scissors. The arytenoid was then removed fairly easily from its attachments with a pair of tissue forceps (see Fig 5).

To facilitate hemostasis, a small right-angle incision was made at the posterior commissure and the flap retracted downward, and all bleeding was stopped. The flap was then sutured into position with plain cat gut sutures (see Fig. 6). The laryngeal cavity was packed with iodoform gauze; the packing

Fig. 7. Appearance of larynx after operation. Notice the external and low position of the left cord.

in the trachea was removed, and the laryngofissure and skin were closed. The packing was not pulled through the cricothyroid membrane.

Since difficulty in swallowing was anticipated, a feeding tube was inserted through the nose.

On the third day, the iodoform packing was removed through the mouth with the aid of a laryngeal mirror and curved laryngeal grasping forceps. No difficulty was encountered.

The patient had some difficulty in swallowing following removal of the feeding tube on the fifth day, but this gradually subsided. After several days of partial occlusion, the tracheotomy tube was removed on the seventh day. No granulations formed, and the mucous membrane healed perfectly.

On the eleventh day, the patient left the hospital, and, up to the present, a year and a half following the operation, has had no difficulty in breathing. Although her voice is very poor, she is able to speak in a hoarse whisper, and she feels that the freedom from dyspnea more than compensates for the poor voice.

Examination of the larynx now shows the left cord in a good external position, and *definitely lower than the right* (see Fig. 7). The lower position of the cord in itself gives considerable airway.

SUMMARY AND CONCLUSIONS.

1. The relief of stenosis in bilateral abductor paralysis of the larynx by making a permanent and adequate airway through the natural air channels has been a very difficult problem.

2. A successful unilateral operation for the treatment of this condition has been presented.

3. This is the first case reported employing the principles suggested by Loré's anatomical study. The results more than fulfilled expectations.

4. Certain modifications were used, but these did not alter the fundamentals of the procedure.

5. The operated cord was not only moved to an external position, but was also maintained on a definitely lower level than the unoperated side. This was an unexpected, but happy, result.

6. In paralyses similar to the one reported, when a physiological operation is impossible or when it has failed, we believe this operation should be the method of choice because of: *a.* The simplicity of the procedure. *b.* The ease of postoperative care and the short duration of convalescence. *c.* The elimination of a bilateral operation. *d.* The assurance of a good airway.

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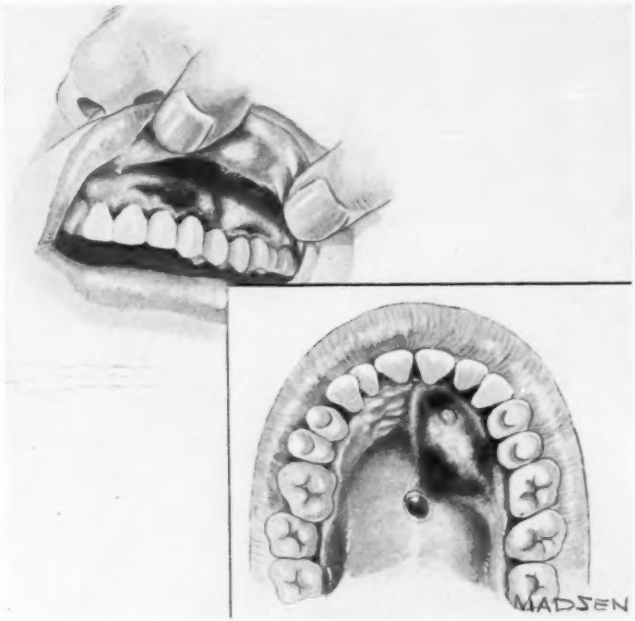
Fitzhugh Building.

MELANO-CARCINOMA OF THE HARD PALATE.

DR. ISIDORE ARONS, New York.

A thorough survey of the literature reveals that melano-carcinoma of the hard palate is a rare disease. Melanotic tumors have been classified variously as melano-carcinoma,



Figs. 1 and 2. Before radiation.

melano-epithelioma, melano-sarcoma, etc. The tumor may arise from either the mucous membrane or the skin. Usually, the only symptom felt is the presence of the tumor itself, but no pain, unless the tumor becomes extensive and inter-

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feres with eating or speaking. When the pigment destroys the blood vessels, it enters the blood stream and emboli of living cells form in a vein, giving rise to generalized metastasis. This condition usually metastasizes early, first in the neighboring lymph nodes, and later as a generalized meta-

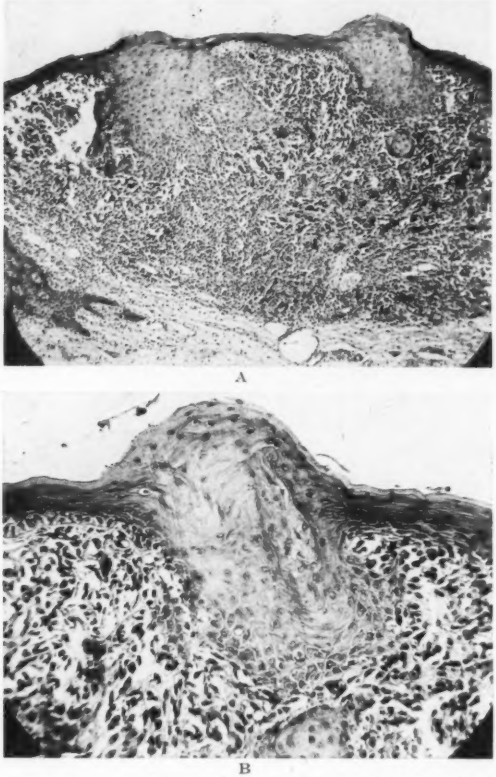

Fig. 3. Melano-carcinoma of hard palate. (a) Low, (b) high.

stasis. Microscopically, these tumors present an alveolar arrangement of spindle and oval cells.

Case History: Mrs. C. C., an Italian housewife, age 52 years, came to my office on June 17, 1937, complaining of two growths on the roof of her mouth, and a discoloration along the gum above the upper left incisor (see Figs. 1 and 2) which had lately been giving her some pain. A biopsy had

been taken at the Skin and Cancer Hospital three weeks previously and the pathological report returned as melanocarcinoma (see Fig. 3).

A cycle of X-ray treatments was outlined, to be given three times weekly, through an intraoral field, size 3 by 3 cm., focused to the anterior upper mandible, using the following factors: 180 KV., 4 M.A., filtration of 0.5 Cu. and 2 Al., 40 cm. distance, 200 Roentgens per treatment; 1,000 Roentgens were given to the two lesions of the hard palate; 1,500 r. of caustic unfiltered radiation to the gum discoloration. A dental mold was made and 62 mg. of radium needles were imbedded in the wax (2 mm. platinum filtration) (see Fig. 4). This mold was placed against the hard palate and remained in

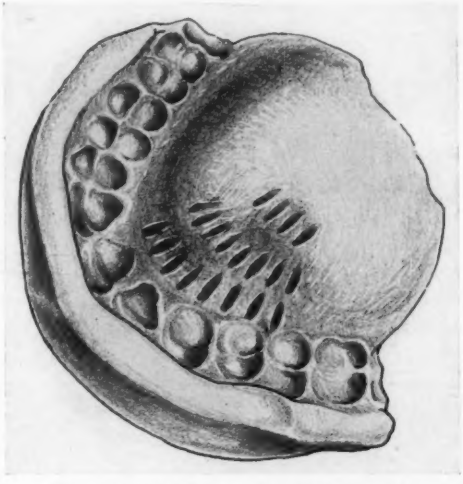

Fig. 4. Dental wax mold. Radium cells.
Dental mold prepared by Dr. George Asch, New York.

place for 50 hours (lead shielding of the tongue) (giving a total dosage of 3,100 mg. hours). A re-examination a week later revealed a good radiation effect (see Fig. 5). It was decided, however, to treat the small lesion at the centre of the palate at 35 cm. distance, using the same factors as previously mentioned. The patient received 600 r. to that area; 1,000 Roentgens of caustic unfiltered radiation were also given to

the upper gum ridge. Because of some pain, a cycle of short wave treatments was outlined, and also a series of liver injections.

The patient went along comfortably until September, 1937, when examination revealed an infection of the treated area.

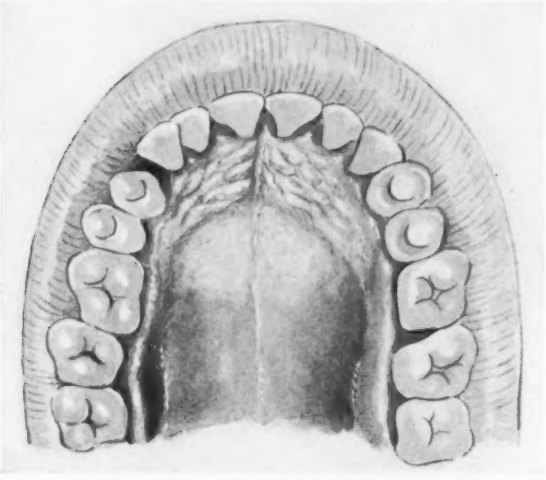

Fig. 5. After radiation.

This was treated by daily irrigations, and the patient was given a second series of liver injections.

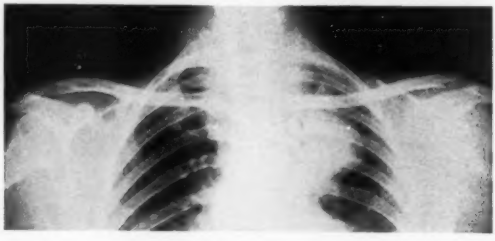

Fig. 6.

On Oct. 16, an X-ray examination of the chest (see Fig. 6) revealed a metastatic involvement of the left hilum. A treatment cycle was outlined, treatments to be given three times

weekly, using the same factors as previously mentioned, field size 10x14 cm., 50 cm. distance, 200 Roentgens per treatment. The patient received only four treatments and was then unable to report further because of increasing weakness. She refused to consider hospitalization and was placed under the care of her family physician.

She later developed metastases to the liver and spine. Her death, on Nov. 6, 1937, was reported by her family physician, five months after her initial appearance at my office.

COMMENTS.

Melano-carcinoma of rapid growth is considered the most malignant type of neoplasm. In most cases, surgical intervention is preferable to radiation therapy. In spite of our carefully outlined program of radiation treatment, the end-result in this case was unsatisfactory. It is evident that either surgical intervention or the application of radium is usually futile. At best, one can produce only a short period of palliation.

57 West 57th Street.

THE CLINICAL SIGNIFICANCE AND THE TREATMENT OF OTOGENOUS FACIAL PARALYSIS.

DR. HANS BRUNNER, Chicago.

Otogenous facial paralysis is surely not frequent, but when it appears it influences definitely the diagnosis and prognosis of the original ear disease. Consequently, it is not sufficient to distinguish between the spontaneous and a postoperative facial paralysis; it is rather necessary to investigate every case as to the cause of the paralysis as far as that is possible. As far as the spontaneous facial paralysis is concerned, we have to deal in first line with acute and chronic purulent otitis.

1. Facial Paralysis in Acute Purulent Otitis: In acute otitis the facial paralysis is rare. Fremel[1] saw, in eight years, only 10 cases. In those cases all depends upon the time when the facial paralysis appeared. Generally we can distinguish between two possibilities: *a.* Facial paralysis appearing in the first days of the acute otitis; *b.* facial paralysis appearing at the end of the third or at the beginning of the fourth week of the acute otitis.

a. There is no doubt that in these cases many young individuals must be included; but I cannot agree that just children show particularly often a facial paralysis in the early stages of an acute otitis, as many textbooks state. In those cases, the facial paralysis as a rule does not appear as suddenly as the rheumatic facial paralysis (Bell's palsy) does (Neumann[2]). We find rather in those cases as a rule some anticipating symptoms, as jerkings of the muscles of the face, disturbances in closing the eyes, etc.; furthermore, the paralysis in those cases does not appear in all branches of the facial nerve simultaneously, but passes over from one branch to the other.

What is the reason for this kind of facial paralysis? We cannot answer this question accurately, since we do not have

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microscopic findings of such cases; but, as usual, we have many hypotheses. Infection of the nerves by contact, compression of the nerve by the hyperemic blood vessels which accompany the nerve, a lymphangitis in the facial canal (Fuchs[3]), toxic paresis of vasomotor nerves and, consequently, disturbances in the nutrition of the nerve (Pollak[4]). All these hypotheses are possible but not proven. The rôle of the congenital openings in the bony facial canal is also hypothetical. It is, however, probable that the rôle of these openings is not of much importance. We know that similar openings in the tegmen tympani are, for the development of intracranial complications, not so important as we have originally believed.

Although the pathogenesis and pathology of facial paralysis in the early stages of acute otitis is not worked out, we know definitely that the prognosis of that kind of facial paralysis is as a rule good with regard to the course of the otitis, as well as the course of the paralysis. Usually the paracentesis is sufficient in order to heal the facial paralysis.

b. Of quite another significance is the facial paralysis in the late stages of acute otitis. Among those cases, the so-called mucosus-otitis is predominant. In those cases we must suppose as a rule an intensive osteitis of the bony facial canal (aqueductus Fallopii), which produces an intensive inflammation of the nerve. Fremel[1] has described a very instructive case of that kind. I have also operated such a case (man, age 28 years, suffering from mucosus-otitis). At operation I have found the nerve exposed for a length of about 6 mm. It is noteworthy that in this case the facial paralysis became evident *after* operation, in spite of the fact that we did not touch the nerve during operation.

The described pathologic changes make it clear that the facial paralysis in the late stage of the acute otitis carries with it a poor prognosis for the paralysis and the otitis. It is an absolute indication for mastoid operation.

2. Facial Paralysis in Chronic Purulent Otitis: Nuehsmann[5] emphasizes that facial paralysis in chronic otitis is more frequent than in acute otitis. On the contrary, Lund[6] points out that he has found in 110 cases of otogenous facial paralysis about the same frequency in acute and chronic otitis. This contradiction can be explained when one considers: *a.* the kind of otitis; *b.* the intensity of the facial paralysis.

a. Every otitis which complicates in any way the internal ear endangers the facial. Since chronic otitis complicates the internal ear far more often than acute otitis, it is evident that facial paralysis is more frequent in chronic than in acute otitis, especially if one counts among the chronic otitis also the cases complicated by a disease of the internal ear; but when we exclude the last-mentioned cases the number of facial paralyses might be the same in acute and in chronic otitis.

b. When we also consider the cases of facial paralysis of very slight degree, there is no doubt that facial paralysis in chronic otitis is much more frequent than in acute otitis. Of

Fig. 1.

course, it needs a very precise observation to perceive a slight facial paralysis in those cases. One sees in these cases only that the nasolabial fold is less prominent on the affected side, or, as Ruttin has pointed out, that the eye on the affected side does not close as firmly as on the healthy side when the head is bent backwards. Very often I have observed in those cases a different innervation of the muscles of the face in rest, as well as in action. Further, I have found differences in the electric irritability on both sides. I wish to emphasize that these cases are very common if one gets accustomed to recognize them. Two cases may exemplify these findings:

Fig. 1 shows a woman, age 50 years, with a chronic otitis on the right side. She had on the right side a total defect of the drum membrane, with many polypi in the middle ear. She was deaf on the right side, and

had a positive fistula symptom. The whole right half of the face is immobile. The right angle of the mouth is just a little bit lower, likewise the right eyebrow; the right nasolabial fold is less prominent. The examination with the Faradic current gives these results:

Irritation of the nerve stem: left, 0.6; right, 0.8. Direct irritation: Forehead, left, 0.4; right, 0.8. Cheek, left, 0.4; right, 0.6. Chin, left, 0.6; right, 0.6.

Examination with the Galvanic current: Irritation of the nerve stem: left, 2.6; right, 2.2. Direct irritation: Forehead, left, 1.6; right, 1.8. Cheek, left, 2.4; right, 3.0. Chin, left, 2.6; right, 2.0.

Fig. 2 shows a woman, age 46 years, with a chronic otitis on the right (cholesteatoma of attic and antrum). Also in that case we find that the right half of the face is slightly immobile, the right nasolabial fold is a little less prominent, the right angle of the mouth dips a little lower, and the wrinkles on the right half of the forehead are less marked. The examination with the Faradic current gives these results:

Irritation of the nerve stem, left, 0.8; right, 1.0. Direct irritation: Forehead, left, 0.6; right, 0.5. Mouth, left, 0.6; right, 1.0. Chin, left, 0.6; right, 0.4.

Examination with the Galvanic current: Irritation of the nerve stem, left, 2.5; right, 3.0. Direct irritation: Forehead, left, 2.0; right, 1.0. Mouth, left, 2.6; right, 2.8. Chin, left, 1.6; right, 2.0.

It is very probable that this slight facial paralysis shows essentially the same pathologic changes as in the intensive facial paralysis in chronic otitis. There is only a difference in degree.

The intensive facial paralysis in chronic otitis will be found: *1.* In cholesteatoma of middle ear, particularly when it is acutely exacerbated; *2.* in chronic otitis combined with a purulent disease of the labyrinth, mostly with circumscribed labyrinthitis; *3.* in otitis media tuberculosa.

In all these cases, the pathologic changes are essentially the same. The facial canal will be partly destroyed, either by nonspecific granulation tissue or by cholesteatoma, or by tuberculous granulation tissue (Podesta[7]). Since the endosteum of the canal is very thick and very resistant, this degree of pathologic change can last very long. It is probable that we have to suppose this degree of pathologic change in cases of slight facial paralysis mentioned above. When the infection progresses, the endosteum will be perforated and the inflammatory tissue lies now between the bony wall of the canal and the neurilemma of the nerve. Instead of the inflammatory tissues, we may sometimes find cholesteatoma in this place (Vermes[8]). Also in that stage the neurilemma resists the inflammation. Consequently, it can happen that the nerve will

be pushed out by the invading granulation tissue and the facial nerve is herniated (Vermes[8]). My former assistant, Schnierer, has described such an herniation of the facial nerve in a case of mucosus otitis. It is noteworthy that this case had, clinically, no facial paralysis. Finally, the nerve succumbs to the inflammation and now the patient will show a facial paralysis. In the facial canal some little abscesses will be found, or a thorough infiltration of the nerve by specific or nonspecific granulation tissue. The nerve thus shows a single or multiple interruption.

It is evident that the prognosis of a facial paralysis of that kind must be doubtful; however, we want to emphasize that

Fig. 2.

such an intensive disease of the facial nerve finally may heal, anatomically and clinically, when we succeed in healing the suppuration within the middle and internal ear. The time for such healing varies, but it lasts always some months; in several cases also some years up to the improvement in the facial disfigurement. Some years ago I found in these healed cases a neuroma of the facial nerve.[9]

In all cases thus far mentioned, the facial paralysis is only a symptom of a suppuration within the middle ear or middle and internal ear; therefore, the treatment of the facial paralysis is included with the treatment of the suppuration within the ear.

3. Postoperative Facial Paralysis: This form of facial paralysis is not frequent, at least today. Neumann[2] counts as postoperative facial paralysis only those cases which show the palsy at once after operation. Koerner[10] reports facial paralysis in 0.9 per cent of cases; Fremel,[1] among 2,634 operations on the ear, only 0.26 per cent. Our experience about agrees with the number mentioned by Koerner. Of course, we note every facial paralysis which appears after operation as postoperative facial paralysis, independent of the time that palsy appears after operation. The reasons for that type of facial paralysis are many.

1. Injury of the Nerve: This is due to an imperfect technique or to abnormal anatomical conditions; they are mostly marked in congenital atresia of the external auditory canal. The prognosis in those cases is as a rule bad; however, a recovery is possible when the nerve is not cut through, or when at operation both ends of the nerve are properly spliced. Neumann[2] has emphasized that facial paralysis is not persistent when the nerve is injured by a sharp instrument.

2. Exposure of the Facial Nerve: This also is due to a wrong technique and very often combined with opening of the horizontal semicircular canal. We must concede that an exposure of the nerve can happen also to a surgeon of great experience; cases with "quiet" cholesteatomata are particularly in danger. More disagreeable yet are those cases in which the facial nerve has been exposed by the suppuration itself without producing a facial paralysis. When such cases are operated, even with the most scrupulous technique, as a rule after operation a facial paralysis, without fault of the surgeon, may ensue. The exposure of the nerve is very often, but not always, followed by a facial paralysis. In this regard, large openings of the facial canal seem to be less dangerous than small openings. The prognosis in those cases is quite uncertain. An amelioration of the palsy occurs by a secondary contraction of the muscles of the face; however, this secondary contraction may appear sometimes after weeks, sometimes after months, sometimes after years. That depends on how much the nerve was damaged, either during operation by sponging, probing or tamponade, or after operation by the postoperative suppuration in the cavity.

3. Hemorrhages in the Facial Canal: This is a hypothe-

sis, which is accepted by many otologists. Also in the last time, Lund[6] accepts this hypothesis. I personally concede that I do not quite understand this hypothesis. First of all, I can imagine a bleeding in the facial canal without opening of the canal only in intensive arteriosclerosis. Further, it is difficult to understand that the facial nerve, whose resistance must always be emphasized, should be paralyzed just by pressure from a hemorrhage. Finally, we have mentioned above that the nerve easily can escape through a congenital dehiscence of the canal when the pressure within the canal is increased. It seems, also, that Neumann[2] does not give credit to such

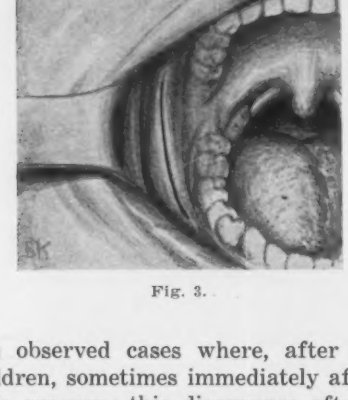

Fig. 3.

bleedings. He observed cases where, after operation performed on children, sometimes immediately after operation a facial paralysis appears; this disappears after two to three hours. Neumann[2] does not attribute this observation to hemorrhage in the facial canal but believes that in those cases the facial nerve, which in children is not thoroughly covered by bone, is damaged by sponging the cavity. We wish to add to this explanation the anatomical fact that in young children the distance between the tip of the mastoid process and the for. stylomastoideum is very small (sometimes only 0.5 cm.); therefore, when in children the tip of the mastoid is roughly removed, a slight injury but sometimes also a more marked injury of the nerve may easily occur.

4. Pressure from the Tamponade: It is evident that a firm tamponade produces retention of secretion which may dam-

age the nerve. That will happen particularly when the nerve is exposed, but it may happen also when the nerve is not exposed. That kind of facial paralysis appears as a rule a few days after operation, and disappears when a good drainage is established.

5. Postoperative Progressive Osteitis in the Operated Cavity: This possibility has been particularly emphasized by Lund.[6] In those cases as a rule a few days after the radical operation (very seldom after simple mastoid operations) a facial paralysis appears, mostly accompanied by labyrinthine symptoms. That type of facial paralysis is produced in such a manner that the osteitis is not checked by the operation and invades the facial canal after operation. Those cases are not frequent. In the microscopic studies which I published many years ago,[11] it has been shown that the osteitis after radical operations as a rule progresses. But this osteitis after operation is mostly mild and subsides in a few days. Only exceptionally is this osteitis rapidly progressive, and may involve the facial nerve, the labyrinth, the meninges or the cerebellum. In the eight cases Lund[6] has described, three patients died of intracranial complications.

This view shows that otogenous facial paralysis generally has a good prognosis, at least as far as the marked disfigurement of the face is concerned. Fremel[1] has found that among 104 cases, only in 5 per cent is the marked disfigurement of the face persistent. Fremel[1] concludes it is thus not necessary to hasten the plastic surgery, especially since the spontaneous contraction usually produces better cosmetic results than the plastic surgery. We surely agree with Fremel[1] as far as the time for the operation is concerned. But there are still 5 per cent of cases that are refractory to conservative treatment and which do not rehabilitate by spontaneous contraction. Unfortunately, Fremel[1] does not state which type of facial paralyses comprise those 5 per cent. In our experience, in this 5 per cent are especially the postoperative facial paralyses. Another group are the cases of facial paralysis after fractures of the base of the skull, and a third group are cases of Bell's palsy. But even in these cases, we first try to get results with conservative methods. If, after one year, no spontaneous contraction develops and no improvement of the electric examination is found, a plastic operation should be performed, for two reasons: *1.* after that time a spontaneous improvement is

hardly to be expected; *2.* after waiting a longer time, the operation is more difficult, at least insofar as the muscle-plug operation is concerned.

Plastic Surgery: Since Halle[12] has recently reported a short summary concerning the different methods, I desire here to describe only my method. It seems that this method is not known, although it was described in 1926.[13] Neither Joseph[14] nor Halle[12] mention this method. Lehmann[15] writes that St.

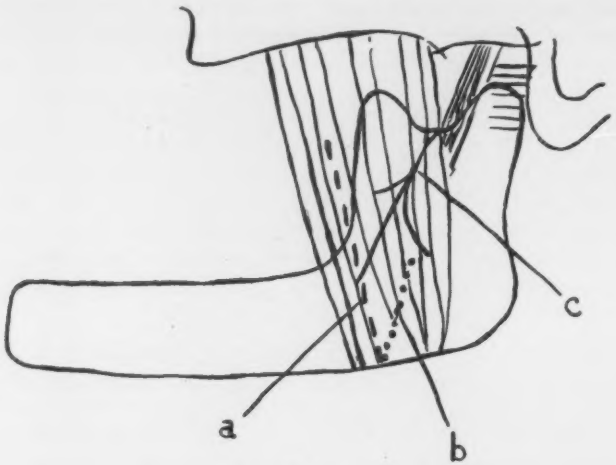

Fig. 4.

Bunnell has been the first who has made the incision for the muscle-plug operation in the mouth. It seems that Lehmann is mistaken. Bunnell was probably the first who has sewn the facial nerve in the Fallopian canal (*Surg.* etc., 45:7, 1927), but as far as I can see he never described either an incision in the mouth or a muscle-plug plastic.

My method has the least risk for the patient and for the surgeon. There is no method for correction of facial paralysis which may be regarded with surety. This also obtains for the method of Duel and Ballance, as Duel and Tickle[16] have conceded. Naturally, this also is true for my method. A method which surely does not furnish ideal results at least should contain the minimum of danger for the patient, as well as for the surgeon. This postulate seems to be accom-

plished by the following method: The operation, which aims to correct only the mouth branch of the facial nerve, was formerly performed in this manner. Exact cleaning and treatment of teeth, anesthesia of the N. mentalis and N. infraorbitalis; infiltration with 0.5 per cent novocaine-adrenalin of the inferior border of mandible, particularly in the region of insertion of the masseteric muscle; brushing of the mucosa of the vestibulum oris with cocaine; incision in the vestibulum oris along the mandible backwards and along the anterior border of masseter, which is palpable upwards (see Fig. 3). Now the masseter muscle will be exposed. This is not always easy when the muscle is atrophic. It is a peculiar finding that

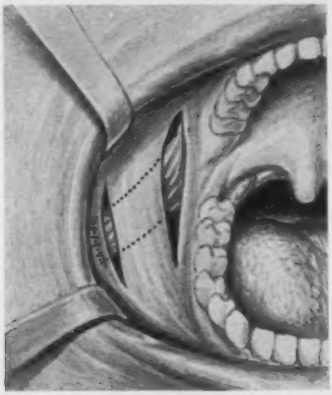

Fig. 5.

in cases of old facial paralysis the masseter gets atrophic on the paralyzed side. Until now, it was not possible to explain this fact. Surely, this atrophy of the masseter cannot be explained by inactivity, since we always innervate the masseter muscles on both sides; however, this experience makes it clear that it is not wise in cases of facial paralysis to postpone the muscle-plug operation for too long a time.

The masseter muscle, now exposed, is then incised. Joseph[14] and the majority of surgeons who do this operation externally emphasize that it is not advisable to incise the masseter as in (a) in Fig. 4, since this kind of incision destroys those branches of the N. massetericus, (c in Fig. 4), which supply the anterior portion of the masseter muscle. Much better is

the incision (b) in Fig. 4, which should not be extended too high upwards, to save the nerve.

When the masseter muscle is very atrophic, it is, of course, not possible to split it. In such cases, I have made use of the whole muscle for implantation without affecting the function of chewing. The inferior insertion, either of the anterior part of the muscle or of the whole muscle, is now cut off from

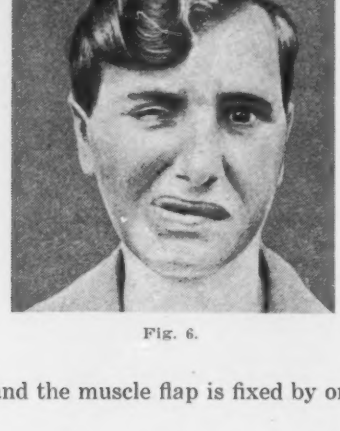

Fig. 6.

the mandible, and the muscle flap is fixed by one or two bridle sutures.

When the muscle flap is prepared, we make an arched incision through the mucous membrane of the mouth near the angle of the mouth, as Fig. 5 shows it. From this anterior incision, the mucous membrane of the cheek is dissected up to the posterior incision. In that way a tunnel in the cheek is effected, and through this tunnel the muscle flap is pulled forward. In the next step of the operation, the posterior incision is drained by an incision in the skin, of about 1 cm. in length, and a rubber drain introduced in the posterior wound pocket. The posterior incision is carefully sutured.

The muscle flap is now anchored to the angle of the mouth. The mucous membrane of the upper and lower lip is dissected up, and in this pocket the flap is fixed. The anterior incision is closed by suture and the angle of the mouth lifted by adhesive strips. I have operated quite a number of cases by this

method. The results have not been ideal, but satisfactory; at least they are as good as the results gained by the other more complicated methods. Figs. 6 and 7 show the results in a girl, age 24 years, who had acquired a complete facial paralysis on the right side after a fracture of the base of the skull in October, 1920. I performed the operation on her in March, 1928. Fig. 6 shows her voluntarily lifting the right angle of

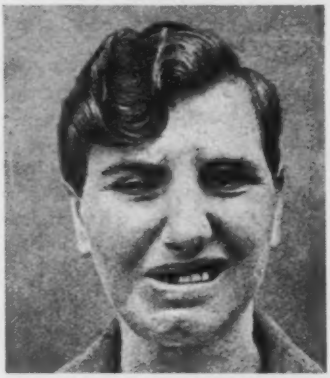

Fig. 7.

the mouth. Fig. 7 demonstrates the teeth in close apposition. The movements of the angles of the mouth are satisfactory. But what we wish to emphasize is the fact that no scar is to be seen on the face. That is the chief advantage of the described method.

Further studies[17] on cases on whom this operation was performed many years ago have yielded some interesting conclusions: *1.* The muscle flap will be resorbed after years. This fact was not formerly recognized because the changes of the flap cannot be controlled by touching in the external operation. On the contrary, it is very easy to touch the flap if the flap is covered only by mucous membrane, as in the endoral method. *2.* The electrical reaction improves, sometimes nearly to normal reaction. These two findings prove that the implanted muscle flap does not work mechanically, but that nerve fibres (from the trigeminus) grow into the paralyzed muscles from the implanted masseter muscle. This fact was pointed out by Rosenthal[18] in 1916. Our experiences sup-

port the observation of Rosenthal.[18] *3*. Although the electrical reaction improves markedly, the voluntary motility of the angle of the mouth will not improve to the same degree, particularly the co-operation of both angles; therefore, as far as the voluntary motility of the angle of the mouth is concerned, it is definite soon after the operation. The improvement of the electrical reaction does not influence the voluntary motility.

These findings have justified me in simplifying the method above described, as shown in the following case:

H. H., male, age 50 years, awoke on March 16, 1936, with a complete facial paralysis on the left side. The patient had a very hard pulse, the

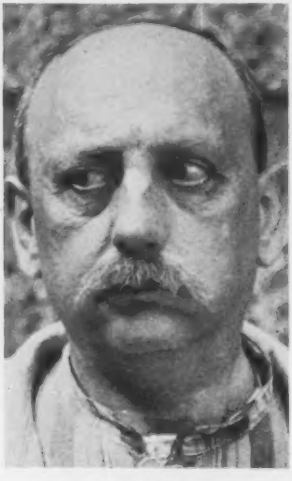

Fig. 8.

Fig. 9.

radial artery had been thickened, and the blood pressure was 220 RR. The electrical examination of the left facial showed a complete degeneration of the nerve.

Diagnosis: Rheumatic(?) facial paralyses. Conservative treatment without result. The operation was performed on May 27, 1936. The masseter muscle was exposed in the manner above described. Profuse bleeding hindered the view. A short flap was dissected from the muscle and anchored with a bridle suture of catgut. The muscle flap was too short to be pulled to the angle of the mouth; therefore, instead of the muscle, the catgut threads were sewed at the angle of the mouth in such manner that the angle remained in over-corrected position. The incisions in the mouth were made as described above. The results of the operation are seen in Figs. 8-11. Fig. 8 shows the patient before the operation; in Fig. 9 after operation. The improvement of the face is evident, particularly in the nasolabial fold. In Figs. 10 and 11, the patient is shown lift-

ing the left angle. In those two pictures the improvement is also evident. The electrical examination in November, 1937, gave normal responses.

In this simplified method, only a short flap of masseter muscle is used. This short flap is pulled forward to the angle of the mouth by catgut suture, or, as I should prefer in future cases, by a strip of fascia lata. This simplified method has two advantages: *1.* The fibres of the masseter muscle will not be diminished to a larger extent. *2.* The duration of the operation will be shortened, because it is not necessary to expose the muscle to its insertion in the mandible. In that way, also, the neighborhood of the external maxillary artery will be safeguarded. This method has the disadvantage that a neu-

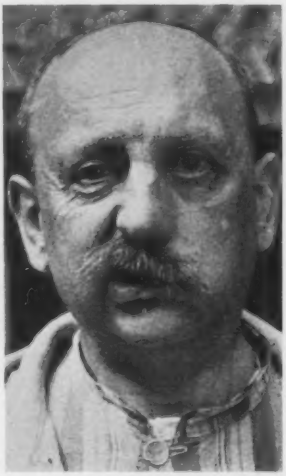

Fig. 10.

Fig. 11.

rotization of the implanted muscle is probably not to be expected.

Summarizing, we may say that the endoral muscle-plug operation, described above, may be recommended. After our experience, the effect of this operation is the following: It accelerates the spontaneous regeneration of the facial paralysis in cases in which a spontaneous regeneration is possible at all. In cases in which a spontaneous regeneration is out of question, it improves the disfigurement of the face, although a normal condition will never be produced.

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PLASTIC SURGERY IN PRAGUE, CZECHO-SLOVAKIA. RECENT EXPERIENCES.*

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The Plastic Institute of Prague, Czecho-Slovakia, is one of the largest, if not the largest, hospital of its kind in the world devoted exclusively to general plastic surgery. It is under the direction of, and was founded by, Prof. Francis Burian, who holds the chair of plastic surgery in the King Charles University of that city. During the World War, Prof. Burian was a Major in charge of the Plastic and Facial Surgery Division in one of the largest military hospitals. Prior to this and during the earlier years of the conflict, he had been a general surgeon. After the cessation of hostilities, he continued to devote himself exclusively to plastic surgery. At this time he founded the Institute of Plastic Surgery, which was then a part of the Czecho-Slovakian Red Cross Service, caring for demobilized officers and men. The constant increase of injuries in civil life and those resulting during the performance of industrial endeavor caused the Minister of Health to transfer the Institute to the Jedlicka Hospital, enlarging its facilities to 36 beds. During the succeeding years the amount of work became larger and the Institute was again transferred, this time to the State Hospital of Prague, and is now housed in a new modernistic building adjoining the general surgical pavilion. The Institute at present has 48 beds, divided into four large male and female wards, and several private rooms. A noteworthy feature is an infants' and children's cribside for the care of pediatric plastic patients, which, for the most part, are of the cleft palate, burn and dermal angiomata variety. The institution has three intercommunicating operating rooms with the most modern equipment available at the present time. In addition to the usual rooms for clinical examinations and

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dressings, there is a room devoted exclusively to photography and prosthetic work, with a full-time technician in charge. Various casts and photographs of the patients are made before, as well as during the several stages of the procedure, and of the final results. Motion picture equipment is also used in recording and reproducing new techniques. There is a separate filing room for the thousands of photographs and a cross index facilitates the finding of photographs of any individual case or of any subject.

The number of operations performed at this institute averages about 900 per year. Of this, 300 to 400 are cleft palates and hare-lip cases. Since Prof. Burian is the attendant plastic surgeon to almost every institution in and about Prague, the amount of material at his disposal is truly tremendous. Operations are performed daily from Monday through Friday throughout the year. The number of operations scheduled per day varies between five and 15 as a general rule. One peculiarity is that no time is taken for lunch and the work goes on continuously from the morning until 4 or 5 o'clock in the afternoon. Prof. Burian himself, however, discontinues operating at noon and leaves the clinic to attend to his private consultations. All operations in the afternoon are performed by his assistants. His first assistant, Dr. Karfik, has been associated with him for about six years, and his second assistant, Dr. Peshkova, an unusually capable young woman, has been associated with him for about three years. The other assistants were mobilized at the frontier during the Sudeten crisis.

The operative routine differs in but few details from the accepted American technique. All operating room linen, including gowns, and gloves which are worn over the usual rubber gloves, are of dark blue or grey linen, for the purpose of reducing ocular fatigue. The use of linen gloves over rubber gloves aids in the handling of tissues. Local anesthesia is the rule, even in young children and infants, although general inhalation anesthesia or avertin is occasionally used.

The local anesthetic is infiltrated in tremendous quantities as judged by our American standards. The tissues are infiltrated until they become markedly swollen and the overlying skin appears stretched almost to the breaking point. From such a procedure one would conceive of a high incidence of

dermal necrosis after operation, but no untoward sequelae were noted during my stay. Local anesthesia was used in resecting a large angioma of the scalp in an infant four months of age. During the procedure one was impressed by the fact that forceps are but rarely used in handling tissues. Fine, small, sharp, curved hooks having one, two, or more prongs are used in place of forceps.

Humped noses and long noses are corrected by using a modified Joseph technique. The large amount of local anesthetic injected into the tissues about the nose distend it to such an extent that it loses all detail of shape and size, appearing like a large potato. Before the soft tissues are elevated from the nasal bones, the nose is shortened. Shortening, however, is not accomplished by resecting a portion of the quadrangular cartilage, but instead the upper borders of both the medial and lateral crura of the alar cartilages are resected on both sides to give the necessary reduction in size, together with elevation of the tip. When a marked reduction in size is necessary the quadrangular cartilage is also resected. Following the subcutaneous elevation of the soft tissues, the removal of the hump is effected by the saw and rasp. Instead of lateral saw cuts and septal pressure in order to infract the nasal bones to the midline and reduce the width of the nose, a heavy forceps is used. In post-traumatic nasal deformity with thick callous formations, the saw, and at times the chisel, is also resorted to. Bone dust and bone fragments are removed with the curette. The bony ridge at the maxilla is removed when necessary by means of a biting forceps. No packing is used inside the nose, but the vestibule, however, is packed lightly with gauze impregnated with antivirus. A small dressing utilizing small rolls at the side of the nose is held in place with adhesive. The pressure dressing with metal splints to which we are accustomed are not used. Postoperative reactions, such as discoloration of the eyelids, etc., were minimum in comparison to that which we observe in this country.

In post-traumatic broad noses with exostoses and deviated septa, Prof. Burian prefers a preliminary septal resection before attempting the plastic procedure. He performs all intranasal procedures without a head miror, using what direct light he might obtain. In these post-traumatic cases, due to the thickness of the bone, the lateral nasal bones are infracted

to the midline by using a lateral saw cut and/or a narrow chisel.

Saddle noses are corrected in the usual manner, with one important addition to the technique. A piece of ivory is introduced into the subcutaneous channel and is permitted to remain *in situ* for one year in order to distend the overlying structures and to break up any scar tissue, thus making a softer bed for the costal cartilage, which is to be introduced about a year later. This technique undoubtedly will tend to obviate or reduce to a minimum the absorption of cartilaginous implants which is believed to be due in some measure to the pressure of the undistended overlying skin and paranasal musculature.

Subcutaneous suppuration when localized is not, as a rule, treated by incision and drainage. Instead, the products of suppuration are aspirated with a needle; the needle is allowed to remain *in situ* and the cavity is filled with bacteriophage or other bacterial antisera. This procedure is repeated whenever the abscessed cavity becomes distended again with pus. This method of treatment in association with cold or iced applications frequently obviates the necessity for incision, thus avoiding consequent scar formation.

Most small nasal facial defects due to the loss of tissue by trauma or disease are repaired by free full thickness skin grafts. It is believed that the well vascularized subdermal adipose tissue and fine connective tissue reticulum as occurs on the face is especially well suited to accept such grafts. Strict attention is given to asepsis, avoidance of trauma to the graft and the bed, perfect hemastasis, safe fixation and mobilization of the graft. Touching of the graft with the fingers is interdicted. The graft is placed in physiological saline solution and moved on a spatula. Hemastasis is accomplished preferably without ligatures. The edges of the flap are adapted accurately to the edges of the defect by a continuous suture. The graft is perforated in several places with a fine sharp lancet in order to prevent accumulation of secretion, thus preventing the graft from floating away from the bed. Compression is secure by utilizing a piece of sponge rubber the exact size and shape of the graft and placed on the surface of the graft which has previously been covered with cellophane. Several sutures that pass through the edges of the

graft and defect are then tied over the rubber prosthesis. This latter procedure accomplishes both compression and fixation. The compress and sutures are removed on the ninth day following the operation and the graft is then subjected to irradiation with a quartz lamp. If the defect is large a pedicled tubular graft or repeated small grafts are used. The percentage of "takes" secured by the above technique is unusually high. Not a single failure occurred during my stay. Strange as it seems, little attention is paid to the color of the graft and the contrast of a defect repaired from the white skin of the abdomen to the surrounding darkened facial skin is quite marked. When this unequal color matching was called to the attention of one of the assistants, he dismissed it as being unimportant.

In the excision of angiomata, scars, keloids and other skin lesions, the resulting defect is closed by extensive undermining of the surrounding skin, or if this is not practical, by full thickness free grafts from the abdomen. The size of the defects and extent of the undermining that enables closure is unusually large. Apposition of the edges of the defect is made by using interrupted subcutaneous sutures. Any resulting bulges are removed by subcutaneous excision of fat and the removal of triangular wedges in the excess skin. A neat, even, flat closure is then secured by a double layer of intracutaneous sutures similar to the Beck mastoid closure. The sutures, however, are brought up to the skin every 4 or 5 cm. If the defects cannot be closed by undermining the skin and if a large amount of scar tissue, periosteum, pericondrium or muscle aponeurosis is present at the bottom of the defect offering a poor base for a free graft, pedicle tube grafts are resorted to. These grafts are obtained from the arm and later directly sutured to the defects after appropriate fixation, or they are brought up from the abdomen by traveling. Instead of migrating the flap by several stages across the abdomen, thorax and neck, the flap is migrated to the arm and thence from the arm directly to the facial area involved.

In superficial defects covered by granulations, Thiersch grafts are employed. The granulations are scraped off and the resulting raw base is cross-hatched with the tip of a sharp scalpel. In addition, little niches are made into the periphery of the defect extending into the skin and are placed about a

millimeter apart around the entire circumference. These niches and cross-hatches liberate contractural scar and permit adequate nourishment of the graft by osmosis. In large and extensive defects small bits of epidermis are pushed into the granulations and serve as centres from which epidermization may spread.

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INFECTION, ARTHRITIS AND ALLERGY.*†

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Without attempting to explain the basic principles involved, the triad, infection, arthritis and allergy is presented as an occasional clinical entity. Its consideration as such in the past has occurred only in scattered instances. Articles using the word "Arthritis" as part of their respective titles have appeared in the *Journal of Allergy* four times in nine years of publications.

In going over 2,684 indexed diagnoses of arthritic, allergic, and asthmatic hospitalized cases (Barnes Hospital, St. Louis, 1926 to 1936, inclusive), one of the 175 cases of acute infectious arthritis and 11 of the 902 cases of chronic infectious arthritis were found with an accompanying diagnosis of allergy.

This first case is summarized, as it typifies developments in the field of allergy and is one of the group of cases to follow:

A. M., white, female, age 30 years, when first seen in September, 1935.

Infectious History: Dates from 1925. Sept. 16, 1925: Acute purulent ethmoiditis diagnosed; Oct. 30, tonsils removed.

1926: All sinuses, except frontals, radically operated.

1927: External frontal operations, right and left; edematous, polypoid membrane found.

1928: Radical antrum operations.

1932: External frontal operations, right and left. Radical antrum operation, right, repeated.

March 7, 1936: Third external frontal operation, right; Oct. 17, fourth external frontal operation, right.

1939: Occasional right frontal headache.

Positive Findings: 1932: First findings of osteomyelitis (ethmoidal) noted; culture positive for hemolytic staphylococcus.

1935: Thick yellow pus noted frequently in right nasofrontal region.

1936: At third right external frontal operation, previously unobliterated ethmoidal cell found in orbital roof. At fourth right external frontal operation, osteomyelitis was found to extend to within 1 cm. of cribriform plate down from involved intersinus partition.

1939: Slight purulent exudate noted in right nasofrontal region at times.

*Read at the meeting of the Ear, Nose and Throat Club of St. Louis, Feb. 16, 1938. One year has elapsed, permitting changes as to the therapeutic results, and an allergic dietary regimen has been added.

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Arthritic History: Dates from 1925. 1925: Multiple joint pains, especially left arm.

1929: Notation of marked pain on motion of left elbow, wrist, and hand; Oct. 10, wrist swollen, sore, next left shoulder, then elbow; Nov. 16, marked pain on motion left elbow, wrist, hand.

1931: Diagnosis changed from acute infectious arthritis to chronic infectious arthritis.

1936 and 1937: Diagnosis: Chronic infectious arthritis.

1938: (Spring) Slight pain proximal interphalangeal joint, right ring finger, and in left shoulder.

1938 (Summer) and 1939: No further symptoms referable to joints.

Positive Findings: 1929: Although the left elbow, wrist and hand were swollen and painful to passive motion, with a temperature of 38° C., there was no X-ray evidence of bone change.

1937: Although there was tenderness and limitation of motion of metacarpophalangeal joints, spine, and left shoulder, there was no periarticular swelling palpable.

1938: (Spring) Metacarpophalangeal joints slightly swollen.

1939: Slight permanent enlargement proximal interphalangeal joint, right ring finger.

Allergic History: Dates from 1908. Feeding difficulty during infancy. Indigestion from milk and eggs during childhood. Before any operative work was started, there were frequent hives, sneezing, watery and mucous nasal and postnasal discharge, and frontal and parietal headache to incapacitation, with nausea or vomiting. Mother has sick headaches, with nausea and vomiting. Sister has hives. Grandmother, maternal, died of asthma.

1938: Occasional headaches, possibly migrainous.

1939: Rare minor allergic symptoms.

Positive Findings: 1929: Boggy nasal mucosa. Skin tests positive for milk and wheat. Nasal smears, though mucopurulent, contain many eosinophiles.

1931: Boggy turbinates. Nasal smears: 75 to 100 per cent eosinophiles.

1935: Mucous membranes of nose pale, especially left, with evidence of polypoid degeneration.

1937: Nasal smears: Large number of eosinophiles in groups, left; polys. predominate on right.

Nasal Culture: Right, heavy growth staphylococcus aureus; left, no growth.

Mucosal Tests: Positive for egg, wheat, chocolate, tomato, carrot, strawberry.

Non-specific Protein Skin Tests: Positive for hemolytic staphylococcus aureus.

Leukopenic Index Tests (by Technician, Oscar Johnson Institute, St. Louis):

6. 9.37	6375	Eggs	8167	6900 6675
6.22.37	5345	Tomato	7900	7435 6325
6.30.37	6700	Wheat	5875	6400 6635

1939: Paleness of nasal mucous membranes.

The leukopenic index test embraces an initial resting fasting total white count, the ingestion of the certain food, and three total white counts at 20 minute intervals thereafter, always using the same pipette for any one test. Its interpretation was based on Vaughan's[1] original observations in 1934, and Rinkel's[2] definition of a compatible curve, as "one having two successive increases in the total white count, the first of

which was greater than the known error in counting, the second being more than 1,000 cells above the initial count, and the count at the end of the hour being definitely greater than the beginning one." This definition was presented in 1936, after Rinkel and Gay[3] in their combined experience had completed over 5,000 tests.

Attention is called to the fact that the diagnosis of arthritis in the case just concluded was based on symptoms and physical findings. X-ray examination was made, but there was no X-ray evidence of bone change. X-ray examination was made in a few of the cases to follow, but the findings were also indeterminate. In other words, diagnoses of "arthritis" have been made without evidence of a granulomatous layer over the cartilage of, or fibrous or bony ankylosis, with atrophy of the bones and tissues adjacent to an involved joint. Many of the joint symptoms and findings may have been due to edema on an allergic basis.

E. F., white, female, age 33 years, when first seen in September, 1937.

Infectious History: Dates from 1926.

1926: Frequent sore throat.

Aug. 21, 1937: Acute tonsillitis; all joint and muscle pains became worse.

Positive Findings: 1937: Tonsils show evidence of active but not acute infection; upper poles are hyperemic, lower poles fibrotic; removed Sept. 17, 1937.

Arthritic History: Dates from 1936. Dec. 1, 1936: Marked joint and muscle pains; loss of 22 lbs. weight.

Aug. 21, 1937: Pains in knees, ankles, arms, forearms, calves, and lumbar region almost unbearable.

Jan. 3, 1938: No further incapacitation.

1939: No joint or muscle symptoms.

Positive Findings: Appearance of almost complete incapacitation because of weakness of lower extremities, with evidence of limitation of motion.

Allergic History: Dates from 1930. Sneezing, postnasal mucous. Seafood causes bloating and extreme illness. Chocolate, tomato juice, causes itching. Milk nauseates, but patient eats ice cream daily; malted milk and cheese every other day. Sandwiches daily. Pork makes patient short of breath.

1938 and 1939: Minor allergic symptoms at rare intervals.

Positive Findings: Posterior tips of turbinates paler than normal. Frontal and maxillary bones and sinuses transmit light clearer than in routine nonallergic patient.

Postnasal Smear: 20 eosinophiles.

Leukopenic Index Tests (by Technician, Deaconess Hospital, St. Louis):

9-11-37	11650	Wheat	9650	6750	5700
9-12-37	6050	Milk	6200	6000	7200
9-13-37	7876	Eggs	6925	6775	6350

G. W., white, female, age 14 years, when first seen in October, 1937.

Infectious History: Dates from 1936. Since 1936, nasal infection, showing pus, with frequent sore throat.

Oct. 30 to Nov. 5, Nov. 5 to Nov. 20, 1937: In St. Louis Children's Hospital with acute ear, sinus and pharyngeal infection.

1939: Infrequent nasal obstruction and discharge.

Positive Findings: After ten bilateral antral irrigations, using local anesthesia, the disintegrating pus changed to negative washings; treatment was also directed to the other sinuses. Infection of lymphoid tissue in pharynx improved.

1939: Occasional hazy mucoid nasal and postnasal discharge containing a few pus cells and a rare eosinophile.

Arthritic History: Dates from 1937. March 1 to hot weather, 1937: Severe arthritis. Feet, fingers were swollen. Heels, balls of feet painful.

Dec. 5, 1937: This was last day that patient awakened with stiff, sore wrists and fingers.

Positive Findings: Not seen during acute attack.

Allergic History: Dates from 1916. Sneezing, alternating nasal obstruction and mucous discharge since childhood. Patient eats excesses of eggs and chicken. Father has pollenosis; mother has long existent colitis.

1939: Infrequent alternating nasal obstruction and discharge.

Positive Findings: Markedly edematous, boggy, grayish nasal mucous membranes.

Nasal Smears: At first, predominance of pus cells with few eosinophiles; later ten eosinophiles to small smear.

Leukopenic Index Tests (by Technician, Office of Dr. O. P. J. Falk, St. Louis):

10-30-37				
10400	Wheat	13600	14250	13750
11- 5-37				
11250	Milk	15900	19950	21200
11-20-37				
9050	Eggs	8150	6750	5250

G. N., white, female, age 30 years, when first seen in July, 1930.

Infectious History: Dates from 1930. Occasional sore throat.

Positive Findings: Darkly hyperemic tonsils; removed 7-10-30.

Arthritic History: Dates from 1930. Jan. 14 to July 10, 1930: Six months of migratory pains in right shoulder, lumbar spine, left ankle, right knee.

Aug. 1, 1930: Marked improvement in all joint and muscle symptoms.

1938 and 1939: No further joint or muscle symptoms.

Positive Findings: Indeterminate when first seen.

Allergic History: Dates from 1929. 1929: Attending physician recorded complaint of tenacious nasal and postnasal mucous.

May 26, to June 4, 1930: Wheezing and cough.

Dec. 25, 1936, to July 3, 1937: Six months of unrelievable cough; attending physician tried all, except allergic, therapeutic measures. There was also sneezing, alternating nasal obstruction, nasal and postnasal mucous discharge. Tomato juice causes patient to vomit. Patient eats pies, pastries, whole wheat bread, wheaties, daily or every other day; thickens gravies and vegetables with flour and cream, and uses eggs excessively in baking.

July 17, 1937: Patient ceased to cough subsequent to dietary regimen.

1939: Occasional wheezing following dietary indiscretions.

Positive Findings: Nasal mucosa, not definitely pale, but surface is fuzzy, and there are pin points of whiteness uniformly attempting to manifest themselves.

Transillumination shows abnormally thin and clear frontal and maxillary sinuses and bones.

Nasal Smears: Preponderance of eosinophiles.

Leukopenic Index Tests (by Technician, George Ives Clinical Laboratory, St. Louis):

7-10-37	7750	Wheat	6750	5900	6300
7-11-37	6800	Eggs	6800	6650	7750
7-12-37	6650	Milk	8300	8400	7450

1939: Slight tenacious postnasal mucous at times, containing few pus cells and a few eosinophiles.

J. K., white, male, age 12 years, when first seen in August, 1937.

Infectious History: Dec. 7, 1936: Streptococcic throat, acute hemorrhagic nephritis, erysipelas, and acute arthritis.

Positive Findings: Aug 24, 1937: Infected lymphoid tissue in each tonsillar fossa removed.

Arthritic History: Dec. 15, 1936, through March 1, 1937: At absolute bed rest with acute arthritis. For four months temperature ranged from 99° to 101° F. Stiffness remains, together with soreness in ankles, knees, mostly right, hips, and fingers at times. Stiffness is more noticeable in mornings.

1938: Extremity symptoms persist in damp, cold weather.

1939: No further symptoms referable to joints or muscles.

Positive Findings: Visible awkwardness and clumsiness of lower extremities. On other occasions, lower extremities seem weak and unable to support patient. Slight tenderness on palpation of right knee, with a little periarticular swelling palpable.

1939: Findings referable to joints and muscles have disappeared.

Allergic History: Dates from 1934. Always sniffling, rubbing or blowing nose, and complaining of alternating nasal obstruction and nasal and postnasal tenacious discharge. Patient ingests wheat, milk and eggs to great excess.

1938: Nasal obstruction and discharge improving.

1939: Infrequent nasal symptoms.

Positive Findings: Nasal mucosa hyperemic.

Nasal Smears: Few pus cells, few eosinophiles.

Leukopenic Index Tests (by Technician, Marshall Browning Hospital, DuQuoin, Ill.):

9-24-37	7000	Wheat	6200	6350	6650
9-25-37	6000	Milk	6400	5500	5600
9-26-37	7900	Eggs	7500	7100	6400

1939: Slight occasional nasal and postnasal mucoid discharge containing few pus cells and a few eosinophiles.

G. F., white, female, age 25 years, when first seen in January, 1938.

Infectious History: Occasional sore throat.

Positive Findings: Tonsils show debris and pus on slight manipulation; removed.

Arthritic History: Dates from 1937. March 26 to hot weather, 1937: Pain and swelling in feet, fingers, heels.

April 1, 1938, and 1939: No further joint or muscle symptoms.

Positive Findings: Indeterminate.

Allergic History: Dates from 1935. Alternating nasal obstruction, mostly at night. Tenacious postnasal mucous. Nose and eyes water a lot. Marked pains in nose and out through cheekbones at frequent intervals; shrinking gives relief. Notices when in house dust, nose bothers for long periods. Patient eats chocolate candy throughout the day; coca-cola several times a day. Sherbet once a day. Sandwiches every noontime.

1938: No further nasal complaints.

Positive Findings: Generally pale nasal mucosa, with slight postnasal secretion.

Postnasal Smear: 8 eosinophiles in a very small amount of mucous.

Transillumination shows almost abnormally clear frontal and maxillary sinuses and bones.

Leukopenic Index Tests (by Technician, Office of Dr. O. P. J. Falk, St. Louis):

2-1-38	5850	Wheat	5900	4450	4200
2-1-38	6650	Milk	7150	8600	10350
2-2-38	5950	Eggs	6400	7200	8750

S. C., white, female, age 47 years, when first seen in February, 1938.

Infectious History: Dates from 1918.

1918: Influenza and sinusitis. Each winter, patient has a "cold" and sinus flare-up.

1939: Sinuses quiescent.

Positive Findings: Feb. 10, 1938: Visible pus from right ethmoidal sinuses.

Feb. 15, 1938: Right displacement irrigation yields pus.

Arthritic History: Dates from 1927.

1927: Pain in right shoulder developed, and during each subsequent winter there have been attacks of marked pain in right arm, right shoulder, right hip, knees and fingers.

1939: First winter without joint or muscle symptoms.

Positive Findings: Feb. 10, 1938: Palpable periarticular swelling metacarpophalangeal joint, right middle digit; limitation of motion.

Feb. 13, 1938: Radiographs show no bone or joint changes.

1939: No positive findings.

Allergic History: Dates from 1918. Nasal obstruction, tenacious postnasal discharge, paroxysms of sneezing. Dietary excess of wheat and milk for years; excessive ingestion of chocolate was causing symptoms in 1936 and was stopped.

1939: No nasal symptoms.

Positive Findings: 1938: Pale nasal mucosa. Transillumination; frontal and maxillary sinuses and bones very clear.

Nasal Smears: Right, pus predominates; left, 8 eosinophiles.

Leukopenic Index Tests (by Technician, St. John's Hospital, St. Louis):

2-13-38	12900	Wheat	10600	10400	10150
2-14-38	9050	Milk	8700	8650	8600
2-15-38	7950	Eggs	10600	9400	9200

C. G., white, male, age 32 years, when first seen in December, 1936.

Infectious History: Dates from 1930.

1930: Occasional sore throat.

1932: Gall bladder and appendix removed.

1936: Three suspected teeth removed.

Positive Findings: 1936: Darkly hyperemic tonsils; removed 12-26-36.

Arthritic History: Dates from 1931. April, 1931: First noticed pain in left shoulder.

Winter, 1936: General migratory pains; started in feet with swelling. Great difficulty in getting about.

Summer, 1937: No swollen joints.

Dec. 15, 1937: Ankles, hand and finger joints became painful and swollen. Vaccines, antigens, diathermy, foreign protein, and physiotherapy have been tried.

1939: Patient reports trouble with feet in cold weather when on them a lot, but patient is carrying on his occupation as representative of one of the drug houses.

Positive Findings: Jan. 10, 1938: Left wrist, and joints of fingers and thumbs, except terminal joints, are swollen and are tender to unusual manipulation. Both ankles are swollen, and there is pain on pressure behind left internal malleolus. Patient walks as if he had flat-irons for feet.

1939: Joint deformities still present.

Allergic History: Dates from 1925. Considerable nasal and tenacious postnasal mucous; alternating nasal obstruction and occasional sneezing.

1939: Difficulty in following any dietary regimen — nasal symptoms have continued.

Positive Findings: Nasal mucosa hyperemic.

Nasal Smears: 10 eosinophiles to a small smear.

Leukopenic Index Tests:

11-30-37	7000	Wheat	5400	5300

(Counts furnished by courtesy of Dr. L. P. Gay, St. Louis—patient did not have time to wait for last count.)

(Remainder of counts by Technician, Office of Dr. O. P. J. Falk, St. Louis):

1-10-37	7700	Eggs	7050	8600	9100
2- 3-38	9050	Milk	9000	7950	7800

H. G., white, female, age 50 years, when first seen in February, 1938.

Infectious History: Dates from 1912. Tonsillitis since childhood.

1920: Tonsils removed; upper teeth removed.

1930: Lower teeth removed.

Feb. 1, 1938: Patient complains of extreme soreness of right side of throat.

Jan. 1, 1939: Sore throat with prostration.

Positive Findings: In upper part of right tonsillar fossa, a pea-sized remnant of lymphoid tissue shows the redness of acute inflammation peripherally with a white point centrally.

1939: Acute pharyngitis subsided after rest in bed.

Arthritic History: Dates from 1927.

1927: Original pain in metacarpophalangeal joint right middle digit.

1930: Severe attack in hands, elbows, hips, knees, and ankles.

1938: After onset of sore throat, severe flare-up in fingers and hips.

1939: No localized joint or muscle symptoms.

Positive Findings: Feb. 8, 1938: First interphalangeal joints of middle digits: pain on and limitation of motion, and palpable periarticular swelling.

1939: Slight enlargement of first interphalangeal joints of middle digits persists.

Allergic History: Dates from 1910. Paroxysms of sneezing. Tenacious postnasal discharge. Pains through face; intermittent swelling of palate; upper plate has been made over many times. Patient eats ten slices of bread daily; pastry every other day. Up to 1936, patient ate 4 pounds of chocolate a week; since then, she nearly becomes frantic when she eats chocolate; it makes her nervous and upsets her mind. For years she has had gastric distress with belching; bloating from ingestion of whipped cream and sweets.

1939: It takes too much character for patient to avoid ingestant allergens. Symptoms, considered the result of angioedema, still make patient miserable at times.

Positive Findings: Always a pale nasal mucosa. Transillumination of frontal and maxillary sinuses and bones show them abnormally clear.

Postnasal Smear: 10 eosinophiles to a small smear.

Leukopenic Index Tests (by Technician, Office of Dr. O. P. J. Falk, St. Louis):

2- 8-38	6650	Wheat	6200 5450	5800
2- 9-38	6000	Eggs	6500 7800	7200
2-12-38	4300	Milk	4500 4600	5400

1939: Definite edema imperceptible.

In addition to the immediate removal of foci of infection, and attempting to eliminate offending inhalant allergens as factors in these patients, an allergic dietary regimen, individual for each case, has been established. The keynote of this diet has been great diversity and no particular foods in continuous succession or excess at any time. The daily intake of sufficient food calories has been insisted upon. Loss of weight in the thinner type of person proved dangerous. Vitamin, calcium, and other indicated therapy was found essential.

It was considered unwise to furnish patients with a typewritten list of foods. Good co-operation has been obtained by making additional suggestions after checking over three day detailed lists of foods ingested. A composite list of foods found permissible in these cases follows. The listed foods in capital letters were recommended at every opportunity, those in parenthesis were to be taken of occasionally, and the others freely.

Fruit

Apple
APPLE SAUCE
APRICOTS
(Banana)
BERRIES, except Straw-
Cantaloupe
Cherries
CURRANTS
DATES
FIGS
GRAPES
Grapefruit
Lemon
Lime
PEACHES
PEARS
PINEAPLE
PLUMS
Tangerine
Watermelon

Cereal

Corn Flakes
Oatmeal
Puffed Rice
Rice Flakes
Rice Krispies

Bread

Cornbread
RY-KRISP (Ralston)
Rye Toast Melba
Pumpernickel

Liquids

Juices (except Orange and Tomato)
Fruit Nectars
(Coffee)
TEA
Water

Vegetables

Asparagus
Beans, Kidney
Beans, Lima
Beans, String
BEETS
Cabbage
CARROTS
Cauliflower
CELERY
CORN
EGG PLANT
HOMINY
LETTUCE
Mushroom
Parsnips
PEAS
(Potato), Sweet, freely
PUMPKIN
Rhubarb
RICE VERY FREELY
Rutabaga
SPINACH
SQUASH
Turnips
Watercress

Miscellaneous

SUGAR
OLIVES
CRANBERRY SAUCE
JAM
JELLY
KARO and MAPLE SYRUP
Raisins
TAPIOCA
JELLO
OLIVE OIL
MAZOLA OIL
Butter
Candy, stick, hard

Soups

Pea
Bean
Vegetable

Meats

(Bacon)
Bass
(Beef, dried)
Beef, Lean
Beef, Roast
Beef, Stew
(Bologna)
(Chicken)
Corned Beef
(Codfish)
Dove
DUCK
Quail
GOOSE
(Haddock)
(Halibut)
(Ham)
Hamburger
(Herring)
(Jack Salmon)
Kidney
LAMB CHOPS
LAMB PATTIES
LAMB ROAST
LAMB STEW
LIVER (Calves)
(Lobster)
(Mackerel)
MUTTON
Oysters
(Pork)
(Pork Chop)
(Salmon)
(Sardines)
(Scallops)
(Shrimp)
Squab
STEAK
TONGUE
Trout
(Tuna)
TURKEY
VEAL STEAK
VEAL STEW
VEAL ROAST

As allergic rhinitis is the initial and simplest form of general allergic disease, the rhinologist is in a better position to recognize allergy in its earliest stages than any one else, and if he permits himself, at times, to assume the role of an internist with otolaryngological tendencies, he may also be the first to recognize arthritis in its earliest stages. Foci of infection will be searched for, removed or treated, an allergic regimen will be established, and clinical results will prove the existence of the triad, infection, arthritis, and allergy.

Summary: The triad, infection, arthritis, and allergy, is presented as an occasional clinical entity.

Conclusions: It is considered that the infection preceded the arthritis, but that allergy was a concomitant factor.

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Beaumont Medical Building.

GUILLOTINE FOR REMOVAL OF HEAD OF MALLEUS IN LEMPERT OPERATION.

DR. D. E. S. WISHART, Toronto.

A preliminary model of this instrument was presented to the American Otological Society on May 5 or 6, 1938.

The principal of the original instrument remains unchanged, but the instrument now illustrated is light, small, efficient and strong.

Fig. 1.

Fig. 1 shows the whole instrument, with scale to show the dimensions.

The second figure shows: *a.* view of the guillotine portion from the top fully open; *b.* from the top partially closed.

The instrument pictured is suitable for operation on the right malleus. The hook is inserted in front of the neck and passes inward, downward and backward around the neck.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 2, 1939.

The hook prevents the tendency of the head of the malleus to disappear in the depth of the attic. The head is severed by closure of the guillotine and is then lifted out with forceps.

The instrument affords two advantages over the shears at present in use: first, there is no rocking of the malleus, so there is no tendency for the handle of the malleus to tear the

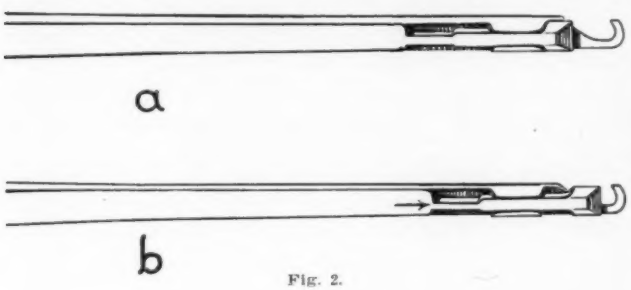

Fig. 2.

membrana tympani; second, the incus is not touched in the procedure and, consequently, its dislocation is avoided.

This instrument is supplied by J. F. Hartz Co., of Toronto.

526 Medical Arts Building.

EDITORIALS

The Editorial Pages of THE LARYNGOSCOPE have been established to offer an opportunity to members of our recently organized Editorial Staff for comments and notes of interest on current events in otolaryngology.

SCIENTIFIC MEDICINE IN DICTATOR COUNTRIES OF EUROPE.

Scientific medicine has suffered in all the dictator nations of Europe. In fact, all sciences have suffered. Germany in particular has declined rapidly. Much of the best German medical research was carried on in the past by Jewish physicians. The new so-called "practical healers," with only two years of training, will not likely contribute anything worth while to science nor will they supply the demand created by the present shortage of physicians. The persecution of Jews in Europe harks back to the dark ages and it is almost unbelievable in this day and age. It has made the Spanish Inquisition seem insignificant by comparison. We must preserve democracy if we are to preserve science. F. R. S.

BOARD STANDARDS.

With the rapid increase in the number of special examining boards, and the large number of well trained specialists already certificated, the best hospitals of the United States and Canada are requiring a certificate for staff positions; however, there are many good hospitals where physicians may treat their patients and do surgery without being required to prove their fitness in a given specialty. This lax attitude on the part of hospital administrators only serves to lower the standard of that particular hospital and lowers the real value of a board certificate. Nearly all teaching hospitals require a certificate. Why not require all hospitals, with a Class A rating, to raise their standards by requiring a certificate from all, except the youngest or associate members?

This will protect the public from those not any too well qualified to diagnose and treat disease. If some hospitals do not want to conform to these standards, let them take a Class B rating. Improvement will come very rapidly under such a system, because all good hospitals want a Class A rating. This will not exclude the general practitioner, but it will require consultations. It will advance preventive medicine and give it the place it deserves in every good hospital. F. R. S.

OTO-LARYNGOLOGIC READING CLUB OF CHICAGO.

Reports of the monthly meetings of this active group will be published regularly in THE LARYNGOSCOPE.

The first Reading Club was organized at the suggestion of Dr. Joseph C. Beck about 10 years ago. At the present time there are five such groups in Chicago and they have been functioning for more than five years.

Dr. Samuel Pearlman and Dr. Jesse Roth described the activities of the first Reading Club in the *Annals of Otology, Rhinology and Laryngology* in 1929, and Dr. Beck went into greater detail on the subject before the Pacific Coast Oto-Ophthalmological Society, at Los Angeles, in 1931.

The Journals reported on are as follows:

1. Journal of Laryngology and Otology (English).
2. Zeitschrift fur Laryngologie, Rhinologie u. Otologie (German).
3. Monatsschrift fur Ohrenheilk. (Austrian-German).
4. Revue de Laryngol., Rhinol., Otol. (French).
5. Acta Oto-Laryngol. (Scandinavian, English, German and French).
6. Archiv. Italia Laryngol. (Italian).
7. Journal, American Medical Association (American).
8. Arch. of Oto-Laryngol. (American).
9. Ann. Otol., Rhinol, and Laryngol. (American).
10. The Laryngoscope (American).
11. Archives f. Ohren, Nasen, u. kehlk. (German).
12. Archives Italia, Tracheo., Broncho., Oesophag. (Italian).
13. Surgery Gynecol. and Obstetr. (American).
14. Valsalva (Italian).
15. Muenchner Med. Wochenschrift (German).
16. Wien. Med. Wochensch. (Austrian-German).
17. Gazette Medicale (French).
18. British Medical Journal (English).
19. Canadian Med. Assoc. Journal (Canadian).
20. Bulletin Johns Hopkins Hospital (American).
21. American Journal of Medical Science (American).
22. American Journal of Pathology and Bacteriology (American).
23. Journal of Anatomy and Physiology (American).
24. Journal of Bio-Chemistry (American).
25. Russki Wrutsch. (Russian).
26. Journal of Pediatrics (American).
27. Rev. D'Oto., Neuro., Ophthalmol. (French).
28. Arch. Neurol. and Psychiatry (American).

29. Tumori (Italian).
30. Journal of Allergy (American).
31. Mayo Clinical Bulletin (American).
32. Transactions of American National Oto-Laryngological and Bronchoscopic Societies.
33. New and Old Books in Oto-Laryngology.
34. American Journal of Surgery (American).

Now that we are sending reports to THE LARYNGOSCOPE at the end of each meeting, the Club decides which one article is most outstanding and merits mentioning; and the reporter prepares it, not with any idea of giving a complete report, the names of the discussers, or the substance of their discussion, but merely a brief resumé as a news item.

It is the hope of your correspondent that men in other places will want to organize such Clubs because I am sure that Medical Reading and the discussion thereof is invaluable.

Not the least of these get-togethers is the social side and reports of interesting oto-laryngological events which took place during the month.

Meetings are adjourned at 10:30 P.M.

J. C. BECK,
Correspondent;
S. A. SCIARRETTA,
Associate Correspondent.

MARCH REPORTS.

Club No. 1. Neue art der Lumbal funktion und neue auwendungs-gebiete derselben. Brunings: *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 44:91, 1938.

Proposes a new method of lumbar puncture by means of a three watt attachment to the needle, each equipped with a petcock so that fluid may be evacuated, siphoned or replaced under pressure. He claims that after enough fluid is removed to reduce the pressure to zero, a further quantity of equal amount can be recovered by siphoning, and still a third quantity of equal amount by inducing a "pneumocranium." The last named process is analagous to pneumothorax, the brain collapsing as a result of a trephine opening and separation of the dura for a sufficient distance beyond the opening.

In meningitis the procedure is to withdraw the three

amounts of spinal fluid, replacing it with normal saline by gravity, and repeating the process several times. The author claims that this method insures a more thorough flushing of the cerebrospinal system than by any other method heretofore employed. Also, he recommends the complete withdrawal of fluid by his method as being helpful in gaining free access to the petrous tip, the Gasserian ganglion and the VIIIth nerve for operative procedures on these structures. He has never seen a case of medullary block result from the procedure.

The Club manifested great interest in the technique, and several members volunteered to investigate it. Inquiry is first being directed to the possibility of acquiring the apparatus, since no illustration of it accompanied the article.

SALINGER.

Club No. 2.

Considerable discussion was caused by the summary of an article on "Mode of Action, Clinical Use and Toxic Manifestations of Sufanilamide," by Long, Bliss and Feinstone, of Johns Hopkins (*Jour. A. M. A.*, Jan. 14, 1939). This paper is a careful study of the drug, both experimentally and clinically, the latter aspect involving 408 patients treated with sulfanilamide. Issue was taken on the rather large percentage of toxic manifestations quoted by the authors, members of the staff of the Illinois Eye and Ear Infirmary stating that this was contrary to their experience. Two otologists with rather large practices gave it as their opinion that the drug is curing many patients with acute otitis media who would otherwise go on to operation. It was agreed, however, that it is dangerous to abandon the watchful attitude hitherto recommended in ear cases because of the fact that the patient seems to be responding to the drug. In this connection, Dr. Joseph Beck cited a case he had seen which seemed to be doing very well under sulfanilamide but, neverthless, showed at operation a completely broken down mastoid.

The abstracter of the article expressed his opinion that while sulfanilamide very often will clear the blood stream organisms of a given type, it is perfectly possible for the original focus to keep on advancing, sometimes with disastrous consequences. In connection with this idea, he cited a case of mastoiditis in which sulfanilamide was administered

when the temperature rose to 104° on the second day; the temperature subsided promptly, and the drug was discontinued after three days. No further complaints were made until a month later, when the patient was admitted again, with a full-blown meningitis, from which he died. Autopsy revealed thrombosis of nearly all the cranial sinuses; it was apparent from studying the specimen that the initial thrombus occurred about the time of the operation, and that the sulfanilamide merely masked symptoms which might have lead to the recognition of the condition. The reviewer also cited a case of chronic osteitis of the superior maxillar from which a pure culture of streptococcus hemolyticus was obtained. Sulfanilamide was of value in controlling the toxic manifestations during an exacerbation but had no effect whatsover, although given in adequate dosage for a considerable period, upon the local condition. It was also brought out during the discussion that the blood content of sulfanilamide (5-15 mg. per 100 cc. of blood is advocated by the authors of the article), and not a customary dosage empirically given, should be the guiding factor. In this connection, a case was cited of a man who received 60 gr. of sulfanilamide over a 24-hour period and turned very cyanotic; the drug was stopped for 24 hours and then resumed for another 24 hours with the same untoward effect. It was then found that he had had kidney disease in childhood and the renal function was defective; the blood examination showed a content of 30 mg. per 100 cc. when the report finally came from the laboratory. S. L. SHAPIRO.

Club No. 3. Clinical Observations on the Pathogenesis and Course of Hematogenous Laryngeal Tuberculosis. J. Spira: *Monatsschr. f Ohrenheilk.*, 72:700, July, 1938.

Spira believes that a hematogenous tuberculosis must be borne in mind when the physical and X-ray findings of the lungs yield negative results, or a hematogenous tuberculosis of the lungs, not greatly advanced, must be considered. From a clinical point of view, he describes two forms of hematogenous tuberculosis of the larynx. The first, a very malignant form, starts with very intensive symptoms at the entrance of the larynx, spreading rapidly into the larynx itself. The second form usually begins at the vocal cords as an infiltration which later undergoes ulcerative changes. This latter form is more benignant and generally heals after a shorter or longer period.

This article is of interest because it may account for some of the so-called "primary" laryngeal tuberculosis cases which have been described in the literature. The discussion, however, brought up the attitude of the laryngologist with regard to treatment. We are now going with a shift of the pendulum from the attitude of open expectancy to a more concerted attack on the problem instead of applying such modalities as heliotherapy, chemical caustics and galvanocautery without any respect for time or place. These methods have definite indications based upon the pathological type of lesion and its site. Surgery, too, with regard to epiglottis amputation, resection of the superior laryngeal nerve and removal of tuberculomas, has also a definite place in the management of laryngeal tuberculosis. Chaulmoogra oil and heliotherapy have not given results that warrant continuing of the use. The latter, especially, may activate the lesion when it is far more desirable to place the larynx completely at rest. While it is recognized that a laryngeal focus in pulmonary tuberculosis places an added burden upon the patient, at the same time it is recognized that these sites may act independently of one another. In other words, that the larynx under proper management may show a healing tendency despite progress of pulmonary lesion, and vice versa.

While these cases of laryngeal tuberculosis are most frequently institutional problems, it is incumbent on the laryngologist to manifest a greater interest in the medical and surgical treatment of laryngeal tuberculosis.

FRANCIS L. LEDERER.

Club No. 4.

Reading Club No. 4 met with eight members present. At the meeting, a paper on "The Prevention of Deafness," by S. J. Crowe and John W. Baylor, *Jour. A. M. A.*, 112:7, was discussed. It was brought out that the authors had studied 15,000 cases of deafness since 1924, using an audiometer with a range of from 32 double vibrations to 16,384 vibrations. During the course of this study, they noticed that high tone deafness seemed often to be due to middle ear obstruction rather than to inner ear lesions. They thought the condition to be due to lymphoid hyperplasia around the Eustachian orifice and that it could only be ascertained in the usual case

with the electric pharyngoscope, and that often a general anesthetic was necessary in this procedure.

They felt that in children, the tubes were often closed by hypersecretion of the mucous glands, and that the first loss in hearing occurred between 10,000 and 16,000 vibrations. The lower end of the scale was not involved except by a slow downward process from 16,000 vibrations. Thus, the deafness was well progressed by the time it became noticeable to the patient. To decrease or destroy hypertrophied lymphoid tissue, X-ray through the jaw portals and radium by means of applicators were advocated.

The condition was found in 60 children, and the authors believed that of the children who were treated before the age of 16 years, hearing would return to approximately the normal level. After this age, less in the way of normal results could be expected.

In the course of the discussion which followed the reading of the abstract, it was brought out that the Rinné test results would be of interest in these cases. It was also brought out that it would be very interesting to know just why Nature makes such strenuous effort to grow lymphoid tissue in this region, especially after it has been removed.

PAUL A. CAMPBELL.

Club No. 5.

The fifth Reading Club met on March 8 with seven members present. The abstract of Oppikofer's article on the question of prolapse of the ventricle of Morgagni, *Arch. f. Ohren., Nasen. u. Kehlkoph.*, 145, Dec., 1938, brought out an active discussion. The author's thesis, that prolapse of the ventricle does not occur on a purely mechanical basis and is, therefore, a misleading diagnosis, met with general support. One additional diagnostic point was brought out; namely, that both a localized inflammatory edema and angioneurotic edema (Quincke's edema) may sometimes produce a clinical picture resembling so-called prolapse and may require to be differentiated.

The discussion of a possible mechanical basis for prolapse brought up the question of laryngocele (laryngeal air sac, or hernia), which condition apparently does occur on a mechan-

ical basis, with probably a predisposing congenital weakness of the saccule of the ventricle. During violent coughing, straining at stool, or strenuous muscular effort, the glottis is closed and expiration prevented by approximation of the ventricular bands, as contrasted to closure of the glottis by the true cords during inspiration, *e.g.*, during hiccoughing. Increased intratracheal pressure during coughing is, therefore, transmitted to the ventricle and saccule. Herniation may take place either externally through the thyrohyoid membrane or internally above the ventricular band, or both. Two such cases were described in the discussion. The occurrence of this latter phenomenon argues against a possible "prolapse" on a mechanical basis. J. R. LINDSAY.

NEW YORK ACADEMY OF MEDICINE

SECTION OF OTOLARYNGOLOGY.

Meeting of Jan. 18, 1939.

The Treatment of Fractures of the Face and Nose. By Dr. Joseph D. Kelly.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOSEPH D. KELLY: To discuss Dr. Whitham's paper is but to praise him for the thorough way in which he has presented this subject to us this evening, and also for the work that he has done in this particular line. It has been my good fortune to have seen some of his cases at the Manhattan Eye, Ear and Throat Hospital, and to have observed some of the results. All plastic work of this type requires a certain amount of originality, surgical skill and judgment, together with a mechanical ingenuity for devising methods and apparatus with which to hold the bones and soft parts in position following traumas. Very often we find that we have not the necessary manufactured article at hand, so it becomes necessary to make it out of what is available. Thus, necessity becomes the mother of some queer looking contraptions.

As Dr. Whitham has stated—and as nearly all will agree who have had the misfortune to be mixed up in this type of surgery—the results in these cases are almost always proportionate to the time of the institution of proper surgical care. The earlier the case is seen and treated, the better will be the result. Inasmuch as many of these cases which we are called to treat happen away from the large cities or city hospitals and are seen and handled for the first time by men not particularly interested in or acquainted with the end-results, many of them which might have been simple adjustment cases shortly after the accident, become complicated and tricky plastic cases later on. It is for this reason that I have advocated for a long time that there should be some one surgeon designated to handle this type of injury in every general hospital, irrespective of its size and location, for with the means of conveyance at our disposal today, airplane or auto accidents are likely to be dumped into a hospital anywhere in the country. This man doesn't have to be a specialist or plastic surgeon—all he has to do is to familiarize himself with what is known about the extent and seriousness of traumatic injuries of the face and nose, and to realize that you can't sew up deep flesh wounds of the face any more than you can sew up deep flesh wounds anywhere else without having trouble—usually resulting in infection and ugly scarring. He should also realize that there are men available to give him advice, if he doesn't want to turn the case over to them for treatment, and also that the best time to do this is as soon as the patient has recovered from shock and it has been determined that there are no intracranial lesions.

Another simple and yet very important procedure which is often overlooked in the general hospital is the care of the skin in those patients who have been dragged along the ground or catapulted from an auto, landing on the side of their face, neck or forehead. The immediate care of such skin with oil solvents, ether, good soap and a mild scrubbing brush, with proper inspections for the first five or six days, and the removal of any stained sloughing area, will most often give a clean looking skin, instead of a spotty, stained area that the patient will carry with him for the rest of his life.

Another place where these lookout men might be of very great help would be in determining the extent of injury in the many children who, from falls and other injuries, have definite fractures which are not diagnosed because they are not seen by the general man until some time after the accident,

when the swelling of the soft parts has become so alarming that the mother thinks it best to bring Johnny to the doctor. The doctor does the best he knows how under the circumstances, but when the swelling subsides, Johnny has a crooked nose, an organized hematoma of the septum preventing proper breathing, or a greenstick fracture which makes itself evident after a year or so. Then things are not so good, and the first one to get the blame is the doctor and the hospital. The problem arises as to what to do with Johnny—whether to let him carry on until he has reached maturity before doing a plastic operation, or whether we should take a chance and operate on him when some young lady wants to know who that crooked-nosed fellow is who lives around the corner. These poor youngsters get an inferiority complex, or beat up all the kids in the neighborhood. I operate on them just before they make up their minds to kill somebody. Sometimes I have to operate a second time to get the result desired. When we think that this might have been avoided, I am ever so strongly in favor of the fact that there should be some arrangement made in every hospital for one of the visiting or assistant visiting men to qualify himself to act at least in an advisory capacity in cases of this type, providing there is no one interested enough to qualify himself for the treatment of them.

When we speak of the time element in these cases, of course we take into consideration the condition of the patient. Should the patient have a fracture of the skull involving the anterior fossae with a fracture of the frontal bones and possibly the cribriform plate, we are not going to straighten his nose, reduce his malar bones, until he stops draining spinal fluid, and then we are not always sure that we are on the right road. I remember one case in particular that we had at Fordham Hospital of an automobile accident in which the patient had a transverse fracture through the bridge of the nose and both orbits, with spinal fluid draining through the nose. We let him alone for 14 days, the spinal fluid drainage stopped, the temperature was within a normal range consistent with his condition, and I decided to transfer him to Manhattan Eye, Ear and Throat Hospital for further observation and repair. Twenty-four hours after transfer—which was carried out in the most careful manner—he began to run a temperature. Twenty-four more and he had a stiff neck and the spinal fluid showed a marked increase in cells. After a meeting of the wise men it was decided to operate. A spectacle incision was made to expose the area in question and on elevation of the soft parts we found a complete separation of the frontal with the perpendicular processes of the superior maxillary, with a fracture and downward displacement of the cribriform plate and a large amount of the right frontal lobe of the brain filling the nasal cavity on the right side. A wide area was exposed over the right side and a large amount of frontal lobe removed until we had a free drainage of spinal fluid and the nasal cavities and opening were kept packed with Dakin's gauze. This patient lived for nine days following this procedure, and I feel sure that if we had prontylin or prontosil to fall back on at that time we might have brought this fellow through. We had two other cases of the same type, but without dislocation of the cribriform plate, and both of them have recovered. One of them was operated upon 13 times before we succeeded in getting her in shape so that she might secure employment.

I agree with everything Dr. Whitham has said, except that if I ever have a depressed fracture of the inferior orbital ridge and malar region, I would like to have him use some wire with a plaster cap rather than pack my antrum with iodoform gauze.

Fractures of the Skull Involving the Temporal Bone. By Dr. J. Winston Fowlkes.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ISIDORE FRIESNER: I am sorry that I had no opportunity to read Dr. Fowlkes' paper, but he presented some facts that are extremely interesting

If you noticed particularly in the second slide that he showed, it evidenced a vertical fracture through the skull, with bilateral deafness, and that has a medicolegal implication in that severe blows to the skull, even those that do not directly involve the petrous pyramid with its contained labyrinth, may be responsible either for total loss of function or at least total loss of the hearing function. I think that is a point that must be remembered in determining the amount of permanent disability that some traumas to the head have caused.

The second point that I would like to discuss briefly is Dr. Fowlkes' statement that these fractures through the pyramid do not heal by bone. I should like to show you one or two slides that illustrate that point.

Slide 1 showed the temporal bone of a man who had an injury to his head—a severe blow to his head—eight years before he developed a fatal pneumonia. The middle ear space, the drum, external auditory canal, short process of the malleus, hammer-head, and tegmen of the inner ear; the tympanic wall and the vestibule, were also shown in this slide. A fracture could be seen in the region that is frequently the site of an otosclerosis. It is this enchondral bone that is always the site of a beginning otosclerosis. Next was noted the stapes, the oval window and the lower pole of the external semicircular canal, which was filled with new formed bone, and changes were shown here. The fracture extended through the labyrinth in this location and was filled with fibrous tissue, but there was no new bone formation, at least not at this site.

Slide 2 presented the vertical section through the vertical portion of the fallopian aqueduct with the facial nerve here, and you see the fracture in this situation extending into the external semicircular canal. This canal is filled with new-formed bone.. You see the same thing in the lower pole of the posterior semicircular canal, and also near the conjoined limb. Now, if the fracture in this situation had torn across the facial nerve, naturally a facial paralysis or paresis would have followed. It might have been followed by such an amount of bleeding in the aqueduct as to cause a compression of the nerve with a destruction of its fibres through compression, again causing paralysis. But you will notice that this fracture is open, and obviously any reinfection of the middle ear would have rendered this individual highly liable to an extension of the infection through this fracture to his epidural space. That is one of the dangers in the fact that these fractures in the region of the petrous pyramid do not heal in their entirety through bone formation. We do see some new bone formation, but the entire fracture is not healed. Here, where it extends into the semicircular canal, the fissure of the fracture still remains open.

In Slide 3 you see very definitely this fracture extending from the tympanum into the enchondral bone that forms the labyrinthine capsule, and into the external semicircular canal. The fracture is absolutely open, with the exception of a small amount of fibrous tissue. The ampullated end of the external semicircular canal and the posterior canal in the region of the conjoined limb are almost completely filled with bone.

The point is this: First, all of these fractures are not necessarily associated with a facial paralysis; secondly, they do not heal by new bone formation. This individual had a discharging ear as far as we know, from a statement in the history which says that the ear discharged subsequent to the fracture, but I do not know whether it was a purulent discharge or simply bloody. Judging from the bony formation in the semicircular canals, I should guess that that was the end-result of a suppurative process.

Osteomyelitis of the Skull. By Dr. Joseph E. J. King.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. E. ROSS FAULKNER: To give credit to a few men who did pioneering work in this field, there is MacKenzie, of London, who first described a

radical method of treating osteomyelitis of the skull. In 1931, Furstenberg published very complete observations on this same subject. Mosher had been doing some work previous to that, and in 1933, he published a very excellent article, in association with Dr. D. K. Judd, the pathologist. In 1936 and 1938, he published further results. In 1937, Dr. King also published quite a series of cases of this disease.

His methods, to my mind, are perhaps a little superior to any of the others. Dr. Furstenberg recommends attacking the diseased bone from the center and working toward the periphery, but this involves the danger of spreading the infection into the sound bone. And it may be a terribly difficult operation—I will never do it again. I did one that way, and I had to transfuse my patient before he got off the table. The vascularity in that case was almost inconceivable. The method of Dr. Mosher is the better one and it is also the better way of stopping the spreading of the disease. It is very difficult to tell where the disease does stop, though Dr. Mosher says that the circumference of the edema is a pretty fair indication.

In regard to Dr. King's remark about Potts' puffy tumor, you must remember that Dr. Potts lived over a 100 years ago. He must have known a little about osteomyelitis of the skull, at that. The only thing to which you can compare Potts' puffy tumor is hematoma of the scalp. In Potts' puffy tumor you will have a swelling which is much harder at the outer zone than at the centre, and that will not contain fluid. There may be a little depression in the center, so that it will have a ring almost like a doughnut in appearance. He described that, and said that it was an indication of a collection of extradural pus beneath the bone.

Mosher quoted somebody—I forget whom—in the statement that if you find pus outside the frontal bone you have certainly got extradural pus. I am a little doubtful as to whether that is true or not. I remember a few years ago I saw a case which was prolonged for a number of months, but which eventually got well, and I doubt very much if he could have had a collection of pus beneath his cranial bones because he recovered. I also saw a very unusual case 15 years ago in Jersey City—a boy with a temperature of 104°, who looked like a very unpromising prospect. We could find no source of any infection, yet the boy had an edematous skull which would pit all over. All I did was to give a bad prognosis. I didn't think anything could be done surgically. There was no apparent infection in his paranasal sinuses. I thought it was purely of hematogenous origin. I could not make out where the original focus was. I went away, leaving rather a hopeless prognosis, and the next day the doctors called up and asked me if I thought there was anybody in New York who knew more about that sort of thing than I did. That was a good many years ago, before I knew Dr. King, and I suggested calling up a general surgeon. I telephoned several and asked them if they had ever seen an idiopathic osteomyelitis involving the bones of the skull. Finally I found a man who said he had seen several. We agreed to meet in the Hudson tunnel and go over there. In 24 hours this boy had changed quite a bit. His general condition was much better. He had a "hot cross bun" skull, and over each parietal region was a nice puffy swelling. The surgeon took the case and made some pretty bold slices—four incisions—and got pus in each case. That boy eventually got well after losing fragments of bone from time to time. There was a case which didn't have an extradural abscess underneath, because the boy made quite a rapid recovery. His final recovery was very much prolonged, but the immediate recovery was rapid.

Relation of Infection to Pneumatization of the Temporal Bones. By Dr. Luzius Ruedi.

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